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THE EFFECTS OF NOISE ON HEARING

By ARAM GLORIG (Los Angeles)*

THE importance of sound to human and animal life is evident throughout the history of the world. Noise and its consequences are by no means peculiar to our generation. Earliest man, and certainly animals, depended upon auditory signals for survival. Hearing and its relation to preservation of life is basic. Even a plan which includes a question of the survival of the human race must include a place for warning signals received through the auditory system. If we examine biblical history we find a good example of the use of noise by one of the early biblical characters when the Walls of Jericho were caused to fall by the blasts of many hundreds of trumpets and shouts from many persons. In the history of the development of civilization, we find that many times the tide of battle was turned because of the shouting of the participants. In the history of our own nation the record shows that attacks made by American Indians were always accompanied by loud yelling designed to terrorize the foe. As recently as the time of World War II noise was used to add to the stress and fear produced by bombing raids.

Although mankind has been associated throughout its history with sound of one kind or another, it was not until relatively recently that examples of the adverse organic effects of sound in the form of noise has been recorded. This fact is not unreasonable since, although isolated examples of noises existed, the widespread noise-exposure that accompanied the onset and development of modern industry was not present. As mass production increased and heavy mechanization progressed the

* Subcommittee on Noise Research Centre, Los Angeles, California.

problem of exposure to noise increased. In spite of this, no recognition of the effects of noise on man were recorded until about 1830 when Fosbrooke of England recorded hearing loss in blacksmiths. Some thirty years later Weber made the first report on hearing loss in boiler makers and railway men. Four years after Weber's report someone who signed himself "volunteer" asked the question, "Can anyone of your readers suggest a remedy for preventing hearing loss caused by rifle shooting?" This question was inserted in *Lancet*, and the answer appeared in a later issue, "Use cotton in the ears". Barr in 1886 and in 1890 published what were then considered complete records of the injurious effects of loud sounds on hearing. Several reports which appeared in the latter part of the 19th century recorded "that railroad workers suffer hearing loss due to noise". Bauer in 1926 first called attention to airplane noise as a factor in hearing loss. In 1937, C. C. Bunch reported a study in the *Laryngoscope* which is probably the first report that includes audiometric data related to noise-exposure. Kryter in 1950 published a monograph called, "The Effects of Noise on Man". This monograph discussed the extant literature on the entire problem very thoroughly.

It was not until about 1946, however, that any concentrated effort was made by the medical profession to study the relations of hearing loss to noise-exposure. At this time, the Committee on Conservation of Hearing, a standing committee of the American Academy of Ophthalmology and Otolaryngology, realized that if they were to discharge their duty properly they must consider the problem of hearing loss and its relation to noise-exposure. With this in mind, a subcommittee was established called, "The Sub-committee on Noise in Industry". This Sub-committee, appointed in 1947, was charged with the specific duty of studying the problems of noise and its relations to hearing loss. Concurrent with the organization of the Sub-committee on Noise in Industry and its research staff, the American Medical Association through its Industrial Health Section held several meetings, notably one in Atlantic City in 1951. A renewed interest in noise control was further aroused by the National Noise Abatement Council. The interest in the medical profession was indicated by the organization of a conference on noise at the University of Michigan. This meeting, held in 1952, was probably the first conference of its kind of a national scope. At the time of this writing the question of noise and its relation to man has become an important national and international problem. It would be difficult indeed, to record here the number of meetings that have been held to discuss this subject. It is probably worthwhile, however, to name a few of the groups that have been organized as a result of the study of noise. Notable is the interest that National Research Council and the Armed Forces have taken in the form of a Committee on Hearing and Bio-Acoustics, known as "CHABA". This committee is responsible for advising the military forces on all facets of the

noise problems. The concern of the military forces is not entirely confined to the organic effects of noise on man. One of the chief problems is the question of community noise and its effect on inhabitants of areas that surround airports and jet testing installations.

Perhaps, one of the most noteworthy developments in the overall problem was the organization of a sub-committee of the American Standards Association designated as the Z24-X-2 exploratory Sub-committee. This sub-committee was organized to investigate the current status of the noise problem. Men of national reputation in the various disciplines were chosen to work with the Chairman, Professor Walter Rosenblith. A few months after its organization a technical aide was chosen to canvass the field for data and write a report reviewing the status of the noise problem at that time. The report was published by the American Standards Association as the Report of the Z24-X-2 Sub-committee entitled, "The Relations of Hearing Loss to Noise-exposure". Many of the data from which the report was prepared were obtained through the co-operation of the Sub-committee on Noise in Industry. The original intent of the organizers of the Z24-X-2 committee was to examine the possibility of setting damage risk criteria for noise-exposure. After a complete review of existing opinions on criteria, and the analysis of the data that had been obtained by the technical aide, Dr. Wayne Rudmose, it was clearly evident that no criteria could yet be established; that a great deal of study was still necessary.

A medical history of this problem would not be complete without mention of the names of men whose interest and energy and drive have exerted a great influence on this problem and its future solution. Through the efforts of such men as Dr. Hallowell Davis, Dr. Howard P. House, Professor Walter A. Rosenblith, Dr. Douglas Wheeler, Dr. Leo Beranek, Dr. Howard Hardy, Dr. Charles Barron, Dr. Clarence Lloyd, Dr. Thrift G. Hanks, Dr. Karl Kryter, and many others, we are able to better understand some of the relations of hearing loss to noise-exposure.

Although the measurement of hearing was possible in an accurate manner prior to 1940, even as early as 1932 and 1933, it was not until 1937 that any actual reports of hearing measurements were made of persons with noise-induced hearing loss. Even at this time it was not possible to measure the sound pressure level of the noise-exposure. Not until the development of the sound level meter and the octave band analyser were these measurements possible. The two principle variables here, hearing and noise, were then measurable and subsequently hundreds of articles were written. When the literature is reviewed, the earliest reports are concerned with the question of hearing loss and its relation to noise. There are, however, two other important parameters of this problem; these are, the effects of noise on behaviour and the effects of noise on communication. Very little was written about either of these before 1940.

The great bulk of reports has been published since then. The study of the noise problem has, by no means, been confined to our own country. A great many pertinent reports appear in foreign journals, particularly in the Scandinavian, Swiss and English countries.

The Legal History

Although the medical history of noise-induced hearing loss is fairly clear cut, and there is no doubt in the minds of anyone that hearing loss will result from noise-exposure, legally the problem is much more complex and not as clear cut. Terms such as disability and occupational disease when used in the legal sense have a meaning entirely different from the medical meaning. For example, the term disability as used in the medical sense indicates a dysfunction of some particular organ or physiological process of the human body. In the legal sense, disability may not mean the same. Within a legal framework loss of function or injury to the human body becomes a disability only if defined as such by law. This is true of the term "occupational disease" also. We may be correct in saying that a loss of function is occupationally induced, but legally that loss of function need not be an occupational disease. What might be classed as an occupational disease or a disability in one state will not necessarily be so classed in another.

As life progressed from the rural community and an isolated family hierarchy to the urban community where occupation was centralized and more or less dominated by single individuals, or small groups of persons, the question of responsibility for occupationally connected bodily injury became increasingly important. In the early history of the industrial revolution when a workman was injured on the job, his only recourse to compensation for this injury was through common law. He could sue in the common law courts only on the basis of negligence, however. If it was established that the employer had been negligent, the employee collected compensation. The significance of this problem is well put by Joseph A. Sullivan, legal advisor to Liberty Mutual Insurance Company in the following words: "Prior to the passage of compensation laws, if an injured worker sued his employer, he would have to have proved that the injury was due to the negligence of the employer in order to recover damages. In addition, the employer had three potent common law defenses: contributory negligence, assumption of risk, and negligence of fellow servants. Many injured workers went uncompensated and the burden of their maintenance, during long weeks of convalescence from injury, fell upon local welfare and charity agencies. It became apparent that the results were too harsh against the claims of injured workers."

It is clear that with such a system there would be many obstacles to payment of compensation for occupationally induced bodily injury. Such a system was bound to result in interminable haggling between employer

and employee, and consequently a system evolved which allowed the employee and the employer to settle these problems on the basis of fixed statutes. These statutes became known as "Workmen's Compensation Laws". Obviously, such a system would suffer a great many growing pains, and many of the problems of Workmen's Compensation are still not settled. The significance of the compensation system was well summarized by the Supreme Court of the State of Washington as early as 1916. The Court stated that "present laws came of a great compromise between employers and employed. Both had suffered under the old system, the employers by heavy judgements of which half was imposing lawyer's booty; the workmen through the old defense of exhaustion in wasteful litigation. Both wanted peace. The master, in exchange for limited liability, was willing to pay on some claims in the future where in the past there had been no liability at all. The servant was willing not only to give up trial by jury, but to accept far less than he had often won in court, provided he was sure to get the small sum without having to fight for it."

Present day concepts have led to considerable confusion as to the original intent of the workmen's compensation laws. At the start, no one had defined the purpose of the workmen's compensation as, specifically, a payment for loss of wages. The interpretations of the laws, however, were such that the principle of "wage loss" became an unwritten precept. Compensation was not made for bodily injury nor for damages but to replace loss of earning capacity. In the early days when suits were filed against the employer on the basis of common law there was no intent to compensate for loss of wages, but to settle for bodily damage with a non-specified sum, the amount of which was unpredictable. When the workmen's compensation laws were passed, the employee was compensated with small specified payments that provided a means of supplementing his reduced earnings. No attempt was made to evaluate the worth of the member that had suffered injury, and although the employee frequently received less than he had if he won against the employer in the common law courts, he was more certain of being compensated as defined by the law. It became not a question of whether the man was compensable, but a question of establishing the connection to his occupation. If the injury was considered to be occupationally induced, the employee received compensation on the basis of a schedule related to the specific injury. The early compensation laws provided only for occupationally related injuries as distinguished from disease. By common agreement a compensable injury must have a specific date of injury and produce a wage loss.

As the progress of industrialization continued and more workmen were employed, it became evident that many industrial processes produced specific diseases as a direct result of occupation. Such conditions

could not be defined as an injury. They did not have a specific date of injury, nor in many cases did they produce a wage loss. As it became more obvious that such conditions were directly caused by occupation, employer and insurance carrier alike foresaw many obstacles to the workmen's compensation system. Unlike injuries, the number of workers contracting occupational diseases could not be predicted from previous records nor was there apparent any means of prevention in a great many instances. On the one hand, occupational disease was thought of as a natural result of the industry. This was part of the risk the man took when he chose to work in this particular type of job. On the other hand, injuries were not particularly peculiar to any one industry. They were thought by employer and employee alike to be a result of fault on someone's part. Whereas occupational disease is insidious in most cases and occurs over a prolonged period, injuries are instantaneous and can be evaluated readily in terms of disability. The question of liability was apparent when an injury occurred. On the other hand, who was liable and how much liability should be attributed to each employer over the prolonged period of onset and course of an occupational disease posed many difficult problems. The matter of accrued liability with reserves for payments became a serious problem with no information as to who and how many employees would contract these diseases. In order to cope with this problem, many of the laws made provision only for specific disease. Frequently, diseases which might be occupationally induced were not considered compensable. Some states went further and limited the accrued liability by providing that all employees, in whom a preexisting condition could be proved, were exempt from compensation benefits.

Most of the recent problems encountered in establishing equitable compensation laws are a result of provisions declaring that occupational disease may be compensable. Noise-induced hearing loss is a good example of this. It is unquestioned that hearing loss reduces a man's capacity to live a normal life, and in some specific instances may reduce his earning capacity; but if the legal criteria used in many states for establishing disability are applied to noise-induced hearing loss, it is not a disability. There is no date of injury nor, in most cases, is there a loss of wages. On the other hand, there is no question that hearing loss can be occupationally induced.

As increasingly more compensation claims were adjudicated the specific provisions of the laws began to assume different meanings because of various and more inclusive interpretations by judges, courts, and compensation commissioners. As time passed and the problem of compensation in general became more important, there was a tendency to broaden the coverage of the statutes. A good example of this trend is found in the interpretation of the law covering noise-induced hearing

loss cases. In 1948 the state of New York declared that noise-induced hearing loss was an occupational disease and involved compensation. The decision was based on a case which proceeded through several courts, and the final decision was rendered by the Supreme Court in favour of the claimant. The man was awarded compensation on the basis of the existing laws. The decision was clearly an interpretation of intent rather than an actual expression of the content of the law. The law at that time clearly stated that there must be a date of injury and a loss of wages before an occupational disease could be considered a disability.

The question was further aggravated by a case in Wisconsin which was filed in 1951. Again, the question of disability was at stake. At that time, Wisconsin law defined disability due to occupational disease on the basis of the scheduled injury law, which stated that there must be a date of injury and a wage loss. The date of injury was defined as the date of the last day of work for the last employer whose employment had caused the disability. The claimant in the Wisconsin case, however, had not lost time at work prior to filing the claim (nor had he lost any since) and one court contended that there could, therefore, be no date of injury. This case progressed through the courts, and the original decision handed down by the Commission was upheld by the Supreme Court. Needless to say, both labour and industry realized that as a result of this decision Wisconsin industry faced a serious problem. How the problem was solved in Wisconsin need not be discussed here. Suffice it to say that a new law written expressly for noise-induced hearing loss was passed and became effective in July of 1955. The law reflects the benefits derived from co-operation between labour, management and medicine. In my opinion, it is the most advanced law in the country today. The Wisconsin amendment to the occupational disease law has been called a law that both labour and industry can live with.

A discussion of the legal aspects of disability would be incomplete without mention of the "compensation for wage loss" concept; the original intent of compensation laws. From the inception of workmen's compensation laws in the decade between 1910 and 1920 until the case in New York state in 1948 there had been no departures from the wage loss concept. However, when the New York claimant received compensation with no date of injury and certainly no wage loss, it was evident that the wage loss concept was in for considerable re-examination. Further, when the state of Wisconsin decided in favour of a claimant under similar conditions loss of wages as a basis for establishing compensation received a serious set back. Heretofore, this concept had acted as a brake which could be depended upon to limit compensation within reasonable bounds. With these interpretations, however, it appeared that industry was in danger of serious financial embarrassment were the wage loss concept completely abandoned.

It is not our prerogative as physicians to discuss disability as defined on the basis of a date of injury and loss of earnings in contrast to social disability which depends upon a man's relation to his total life. Much of what we might say would be biased. As physicians it is our duty to preserve man's various functions and restore them whenever possible. As doctors we cannot and should not relate loss of function to dollars and cents. We must evaluate loss of function by the effect on the total man. On the other hand, we are members of the community, and are in this capacity concerned with loss of function as it affects the community, also. What the community can afford to pay for loss of bodily function and whether payment shall be based upon man's relation to his industrial life or to his total life must be regulated by community opinion. As members of the community we might argue that compensation benefits must be based upon the opinions of the whole community not only the industrial community. After all, the employee is a member of the industrial community relatively for only a small part of his lifetime; but he remains a part of the total community for his entire life. As physicians we must urge that the industrial community do everything possible to preserve human functions. We should be impartial in our decisions regarding all members of the industrial community, which includes employee and employer alike. Our concern is conservation not compensation. Much of what we do will influence the results of the application of compensation laws to employer and employee; but whether the laws are based on wage loss or social loss must be decided upon primarily by the total community.

Noise-exposure

One of the most difficult problems associated with a study of noise-exposure is that of devising a method of describing and categorizing in a meaningful way the multitudinous types of noise-exposure encountered. In order to describe noise-exposure adequately for correlation purposes, some numerical method of classification must be evolved. This method of classification must provide a way of combining the measurement of quantity of sound energy with measurements of its time distribution. The result might be called a "noise-exposure-index". One approach to the total problem would be to index noise-exposure first with respect to its *physical dimensions*, and then as a further step, to index the effects of the noise-exposure on man. The problem of assessing noise-exposure is very complex, and large amounts of data must be gathered and analysed before we can say what a practical and useful assessment of noise-exposure is.

Recent measurements indicate that sufficiently detailed information about the energy content of noise-exposure is not provided by RMS measurements. A report from Harold Crouch of Eastman Kodak Co., shows that although the RMS values of overall levels of certain so-called

"steady state" noises were in the region of 100 db., the instantaneous levels were as high as 120 db. for 10 per cent. or 15 per cent. of the time. In my opinion, evidence such as this must not be overlooked. We may find that peak factors in noise-exposure correlate better with resulting hearing loss than do some of the factors we have already considered, such as the frequency distribution of energy in the noise-exposure. Figs. 1 and 2.

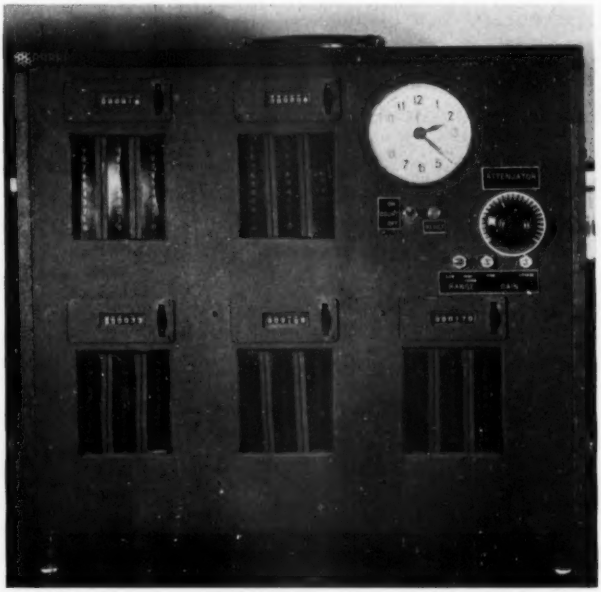

FIG. 1.
Noise-Time Integrator.

In spite of the variability of the industrial processes and of the accompanying noise-exposures, large numbers of industrial audiograms show the same type of hearing loss: the loss begins in the region of 4,000 cps and spreads in both directions as exposure time increases. It appears then, that energy content of noise, its peaks, its time distribution and its total duration, regardless of spectral distribution, are factors which bear close scrutiny.

The Research Centre of the Sub-committee on Noise has recently undertaken a programme of gathering and analysing detailed information about noise-exposures. The first goal of this programme is to devise a method of indexing noise-exposures from measurements of their physical parameters; the second goal, which I feel will not be realized for some

years, will be to index the auditory effects of various noise-exposures on man.

The relations of noise-exposure to hearing loss are not well understood. We do know, however, that there are four major factors of noise-exposure that are important to the production of hearing loss. These four factors are:

1. The overall noise level.
2. The frequency composition or spectrum of the noise.
3. The duration and time distribution of the noise-exposure during a typical work day, and
4. The total duration of noise-exposure during an expected work life.

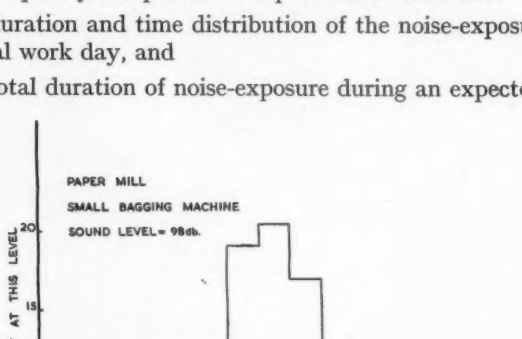

FIG. 2.

Our research has emphasized the importance of the relation of noise and time, that is, the importance of noise-exposure. Distribution of exposure time and total time of exposure are important integral parts of the noise problem. It is important to know whether the daily exposure has been continuous or intermittent. Intermittent exposures appear to produce less hearing loss than continuous exposure of the same total time even though the intermittent exposures are accompanied by considerably higher overall noise levels. This apparent effect, if it proves to be real has two important implications: (1) a statement of the time distribution of exposure must accompany the description of any noise-exposure; (2) intermittency of exposure may be a method of ear protection. Some support for this concept may be found in our study of flight line jet engine mechanics. A recent analysis of auditory records of 175 flight line

jet mechanics and 35 employees of a container manufacturing company, all between 26 and 35 years of age, showed 18 db. more loss at 4,000 cycles in the median audiograms of the container fabricators. Also, 75 jet mechanics between 36 and 45 years of age showed as much as 25 db. less loss at 4,000 cycles than did 45 additional container fabricators in the same age group. We recognize that several unknown factors here may have influenced the results. Perhaps the most important of these is the difference in the use of ear protection. Most jet engine mechanics wear ear protection faithfully; the acceptance among container fabricators, on the other hand, is not as good. In spite of this difference I feel that one of the reasons why jet mechanics show less loss, is the time distribution of exposure. Consider the fact that jet mechanics work in a noise level at least 25 db. higher than the noise level in the container fabricator factory: even when ear protection is worn the noise level at the jet mechanics' inner ear is still about 10 db. higher than the noise level to which the container fabricators are subjected. The container fabricators in the sample were exposed to a continuous noise of overall level equal to 102 db. The jet mechanics were exposed intermittently for varying times to noise levels of 130 to 140 db. Although the overall levels differ by 30 to 40 decibels the spectra of these two noises are not significantly different. It seems reasonable then to assume that the significant difference between the two noise-exposures is the time distribution of the exposure.

Relations such as those shown in the above discussion apply only to continuous type steady state noise. We know too little of impact noises and the relationship of peak factors in noise. When discussing relatively steady state continuous noises, however, it is safe to say that there is a distinct time-energy relation. That is to say, as energy is increased time must be decreased and vice versa. These relations are true only if the noise and exposure time are continuous. I do not feel that continuous exposure will produce the same effect as when the exposure is interrupted by rest periods. For example, the effects of four hours of continuous exposure to 110 db. are considerably different than those from eight hours of exposure to the same level with varied rest periods between the exposure periods, even though the total energy may be the same in both cases. The response of the ear to the exposure is not the same in both cases. The ear that has had a chance to rest between exposures is probably more resistant to permanent loss than the ear that has been exposed continuously without intermittent rest periods.

Effects of Noise-exposure

The known effects of noise-exposure are of two types: *non-auditory* effects which cause interference with communication by speech or which otherwise influence behaviour and *auditory* effects, which consist of

temporary hearing loss and permanent hearing loss. Consider first the non-auditory effects of noise-exposure.

Non-auditory Effects

Speech Interference. Perhaps the best understood non-auditory effect is interference with communication by speech. Noises that are not intense enough to cause a hearing loss may interfere seriously with communication by speech. The average of the sound pressure levels in three octave bands, 600 to 1,200 cps, 1,200 to 2,400 cps, and 2,400 to 4,800 cps is a good indicator of the amount of interference that noise will have on speech communication. This average sound pressure level is called the Speech Interference Level. If you wish to converse in normal tones with someone who is five or six feet away from you the average sound pressure level in the three bands must not exceed 50 db. If you are willing to shout, the sound pressure level can be as high as 68 or 69 db. If you are willing to converse in a shout at a distance of no more than 6 inches the Speech Interference Level can be as high as 90 db. Note this last number. Noises with this average sound pressure level in the three octave bands may be potentially damaging to hearing. In fact, a rough way of detecting hazardous noise-exposure is to determine whether or not one must shout at close range to communicate by speech.

Annoyance. Perhaps the most talked about non-auditory effect of noise-exposure is annoyance. It is a common error to equate the annoyance produced to the potential menace to health that might result from exposure to the noise. There is no necessary connection between annoyance and potential damage, or between lack of annoyance and lack of hazard. Actually certain well deplored pastimes of the human race attest to the fact that the pleasantness of a stimulus is not necessarily an indication of its beneficial effects on health.

The annoyance caused by noise is largely a psychological response. Noises made by neighbours and by their dogs tend to be more annoying than the same noises made by members of our own households. The annoyance caused by aircraft noise is intensified by the fear that the aircraft may crash onto us. It is especially annoying to have to listen to speech that is just too faint to be understood.

How can we measure annoyance? One way is to check complaints. If the noise brought forth a law suit it was indeed annoying to someone. There are other less drastic but perhaps less realistic methods for rating annoyance. We can conduct opinion polls and ask community members if such and such a noise is annoying, and we can reproduce noises in the laboratory and ask subjects to rate them according to annoyance. None of these methods is really satisfactory. In general higher intensity, higher frequency (above 1,500 cps) and intermittency tend to increase annoyance, but there are large individual differences in reaction. Annoyance

itself is not a health hazard and the fact that a noise is annoying does not establish it as a damage risk.

Efficiency. A third non-auditory effect of noise-exposure is possible loss of efficiency. Efficiency in performing mental and motor tasks under various conditions of noise-exposure has been much studied because of its practical importance. A major weakness in the design of experiments, however, has tended to invalidate most of the studies conducted in industry. This weakness is the failure to control changes in the non-noise-related environment. Frequently when the noise environment changes it is because there has been a move to a new building or a change in process accompanied by changes in work tasks, in temperature, in lighting, etc. Changes in efficiency are known to be caused by any one of these non-noise changes in environment. Other difficulties with industrial tests of efficiency are that the numbers of employees participating in the studies have been too small to average out the effects of individual differences or even to allow us to estimate statistically what effect these differences might have on the results. Further, a psychological factor enters here. It has been found that the performance made on first encounter with a test seems to "persist" even through environmental changes. This means that if an employee did poorly on a test given to him first in noise, he would tend to do poorly on subsequent administrations of the test, even in quiet—and vice versa.

The conclusions drawn from the most reliable tests of efficiency in industrial noise are that unfamilar noise temporarily reduces efficiency, but that when an employee becomes accustomed to the noise his efficiency returns to a non-noise performance level.

Physiological Changes. Certain temporary physiological changes also occur in the human body as a direct result of noise-exposure. The usual emergency reactions of the body occur in response to sudden sharp sounds such as gun fire. There may be a sharp muscle contraction over the entire body. The breathing rate may increase. Blood pressure rises. The heart rate increases. There is an increase in sweating, etc. It is possible that a frequent recurrence of these changes might impair health. Fortunately, the magnitude of these physiological responses wears off rapidly with repetition of the noise-exposure.

About the non-auditory effects of noise, we can say in summary:

1. The interference of noise with speech communication under a variety of conditions is well understood and can be predicted from measurements of the sound pressure level in certain octave bands of the noise.
2. The annoyance caused by noise is a highly individual phenomenon and as such is not easily predicted or measured. The fact that a noise produces annoyance does not mean that it is bad for health.

3. Efficiency in the performance of mental and motor tasks in industrial settings is only temporarily affected by exposure to unfamiliar or intermittent noises.
4. Physiological changes resulting from noise-exposure lessen with repetition of the exposure. At typical industrial noise levels it appears that no unhealthy physiological reaction results even from prolonged exposures.

Auditory Effects

Temporary Hearing Loss. Our studies of the temporary auditory threshold shift caused by noise-exposure have clearly demonstrated several important facts related both to temporary threshold shifts and to permanent threshold shifts. Some of these facts are:

1. The temporary elevation of auditory threshold which results from one day's exposure to noise levels of 100 db. or more may vary from no shift to 40 db.
2. Exposure to typical industrial noise produces the largest temporary loss at 4,000 and 6,000 cycles per second.
3. The major portion of the temporary loss occurs during the first one or two hours of exposure.
4. The amount of temporary loss is roughly the same for the same person from day to day but it varies from person to person according to a normal statistical distribution; that is, few persons have very large or very small shifts; and most of the losses cluster around an average value mid-way between the large and small shifts.
5. The amount of temporary loss and its frequency location vary with the amount and frequency location of the permanent loss; the more the permanent loss at any frequency the less the temporary loss at that frequency.
6. Recovery from temporary or transient hearing loss occurs mostly within the first hour or two after the noise-exposure has ended.

A complete long term study on the onset of temporary threshold shift and the recovery from temporary threshold shift is being conducted at our laboratories. We feel that there is a distinct relationship between temporary hearing loss and permanent hearing loss. There are a few things that can be said about temporary threshold shift with considerable confidence. For example, a noise which does not produce a temporary hearing loss will not produce a permanent hearing loss; that hearing loss produced by a specified exposure over a long period will probably not be greater than the temporary hearing loss produced by a specific short term noise-exposure; that the configuration of the audiogram seen in the temporary threshold shift will resemble the audiogram that

will be found when the permanent hearing loss is measured. Obviously, these assumptions are not based on fact since the relationship between temporary loss and permanent loss has not been validated. Such a validation will be obtained from the long term studies of serial audiograms on persons in whom temporary threshold shift studies have been

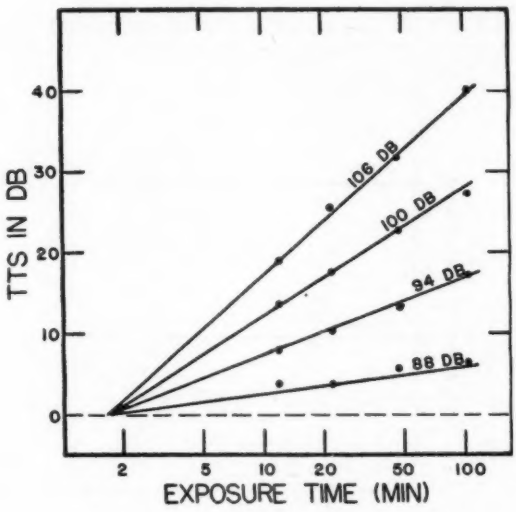

FIG. 3.

made. It is not unreasonable, however, to assume that the temporary threshold shift is a reflection of the long term process, and that some answers may be derived from the study of the temporary hearing loss produced by a specified noise-exposure.

Much of the earlier work on temporary threshold shift was done with pure tones and the relations of pure tones to broad band noise is clearly not understood. Our research is based on the use of broad band noises such as flat spectrum white noise (Fig. 3) and various bands taken from white noise (Fig. 4). In this manner we can establish a relationship between octave band and total noise which may produce a regression equation enabling us to predict by a weighting formula what various industrial noises will do as a function of temporary threshold shift.

The temporary threshold shift has other important parameters. Some of these are (1) its relation to industrial audiometry. We have stated previously that as much as 35 to 40 db. of shift may be sustained during one work day. Obviously, if an audiogram is made at the end of the work day prior to a rest period, this amount of temporary loss will be included

and the audiogram obtained will not be a true indication of the permanent loss. Unfortunately, all audiograms, particularly follow-up audiograms, cannot be made prior to any noise-exposure. It would be ideal to be able to do all audiograms in the morning prior to starting work. This is not practical, however, and therefore, some other method of preventing inclusion of the temporary loss when measuring permanent loss must be devised.

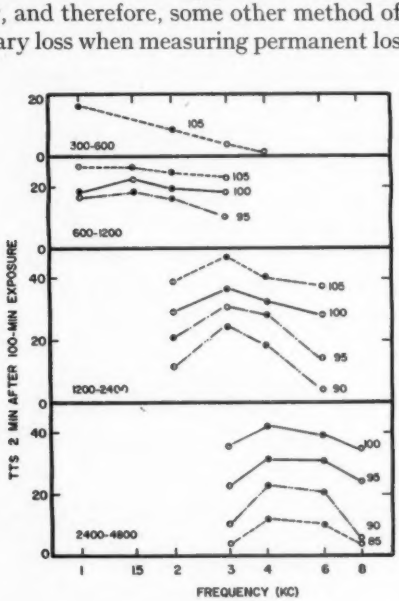

FIG. 4.

So far, our research indicates that if an audiogram is made thirty minutes after leaving the noise much of the temporary threshold shift will have disappeared. (2) The effect of permanent loss. Preliminary data indicate that there is definite relationship between the amount of permanent loss present and the amount of temporary threshold shift. As permanent loss increases, the amount of temporary threshold shift decreases (Fig. 5). This relationship is almost linear up to a point where if permanent loss is enough there is no longer any temporary threshold shift. (3) Its relation to susceptibility. Many tests for susceptibility to noise-induced hearing loss have been based upon the amount of temporary threshold shift produced by short exposures to various types of sound. A study of these proposed tests for detecting highly susceptible ears has shown ample evidence that there is no test which will predict susceptibility to noise-induced hearing loss of any degree. Despite this, we are continuing the search for a susceptibility test as a secondary product of our studies on temporary threshold shift.

Let us consider for a moment the problem of susceptibility to noise-induced hearing loss. Much of the fear of the industrial noise problem has been based upon the supposition that a great many persons would be highly susceptible to noise-induced hearing loss. From our studies of several thousand records, we feel that susceptibility follows a normal statistical distribution; that few persons, therefore, are highly susceptible

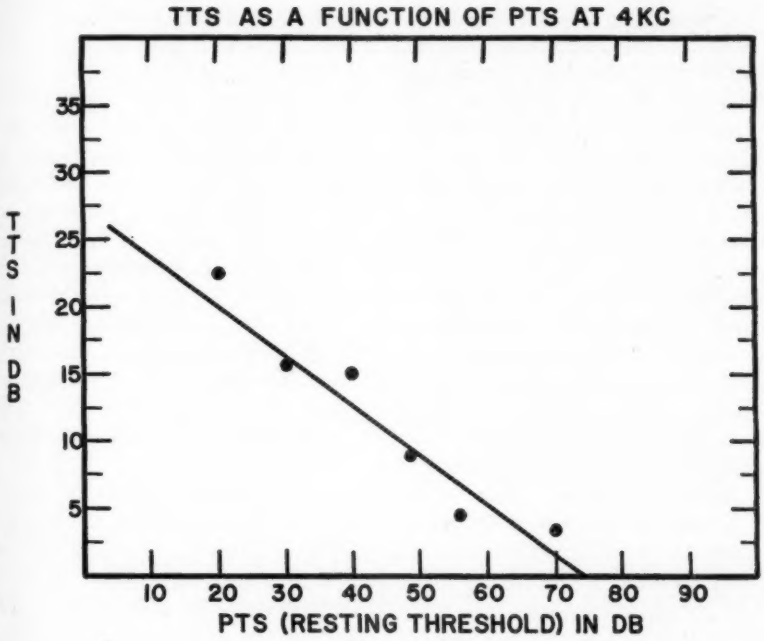

FIG. 5.

to noise-induced hearing loss. An examination of the literature shows a complete lack of documented cases of sudden severe hearing loss due to continuous noise-exposure. In my own experience, I have seen a few cases which were probably the result of explosive blasts such as gunfire, etc., but I have never seen a case where continuous noise-exposure has produced a severe sudden hearing loss. Undoubtedly, there are a few such persons, but these will be discovered by a hearing conservation programme where monitoring audiometry is carried out in the form of follow-up hearing tests.

Permanent Hearing Loss. Long continued exposure to intense noise can produce permanent hearing loss but the process of production is now well understood. The end result, namely the pathology of permanent

noise-induced hearing loss, has been demonstrated in animal ears. Noise-exposure produces an inner-ear damage which may vary from minor changes in the hair cell endings to complete destruction of the organ of Corti. The mechanism of the production of this pathology is not completely known but it appears that over-stimulation by noise for long periods of time produces a metabolic change in the cell which in turn causes degenerative damage to the cell structure.

The specific causal relations of hearing loss to noise-exposure are very complex and many questions must be answered before definite statements can be made about how much hearing loss will result from a given noise-exposure. Investigations so far have produced three particularly important points about permanent hearing loss. First, serial audiograms of persons exposed to intense noise show a characteristic progression of hearing loss. Early losses appear at frequencies between 3,000 and 6,000 cps. Usually losses appear first at 4,000 cps and then in time spread in both directions until hearing for most of the audible frequencies is effected. The extent of the spread depends upon the amount of noise-exposure sustained. Impairment of hearing is usually not noticed until losses at the speech frequencies, 500, 1,000 and 2,000 cps are 15 db. or more. Substantial losses may occur at frequencies from 3,000 cps to 6,000 cps without producing any subjective awareness of changes in hearing. The production of noise-induced hearing loss is a slow and insidious process and years of exposure may elapse before any significant noticeable loss of hearing occurs.

Second, we cannot directly equate the deleterious effects of noise-exposure and the energy content of the noise. That is, double the energy content in the noise does not produce double the hearing loss. We assume that the larger the total energy content of the noise the smaller the time of exposure required to produce the same amount of hearing loss, but the exact relation between time and energy is not known.

Third, the total amount of hearing loss produced by noise-exposure depends on many more variables than was thought originally. Hearing loss varies with the type of exposure and its degree of intermittency, with the individual exposed, the total duration of the exposure, and with the degree of consistency of use of ear protection.

Noise Exposure and Permanent Hearing Loss. Throughout the discussion we have referred to the hearing loss produced by noise-exposure, not by noise. The concept of exposure, of the need for time of development of hearing loss is important to the physician. Brief exposures to most industrial noises will produce no illness or damage to the ear. Whenever the causal relations of hearing loss to noise are considered the physician should consider each of the four factors bearing on noise-exposure:

(1) The overall noise level to which the man was exposed.

(2) The frequency composition or spectrum of this noise, probably as expressed in octave band sound pressure levels.

(3) The duration and time distribution of noise during a typical work day.

(4) The total duration of the noise-exposure expected during the work life.

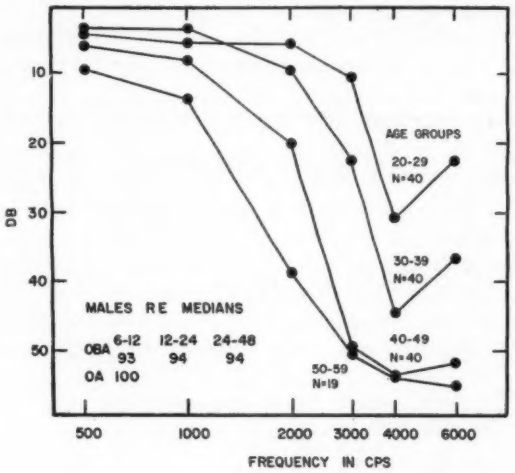

FIG. 6.

To elucidate these last two factors consider the following.

The physician is frequently confronted by persons who work in high level noises, perhaps as high as 130 or 140 db. Before drawing any conclusions about the effects of exposure to these noises the physician must inquire carefully about the length of time spent in the noise. Brief exposures to these noise levels will not produce severe hearing loss nor in most cases even significant hearing loss. On the other hand, the physician also encounters persons who work in lower noise levels, perhaps 100 db., and again exposure time is important. The possible effects of extended exposure to these lower levels must not be minimized. In many instances persons who are exposed to the 100 db. noise have much longer total duration of exposure than do the men who work in excessively high noise levels. If the noise level is approximately 100 db. and the exposure has been long, existing hearing loss may be directly related to the noise-exposure (Fig. 6).

Some industrial noises will produce significant hearing loss only if the exposure continues over 15 or 20 years. Many operations that are associated with these industrial noises, however, are characterized by rapid

job turnover. The total duration of exposure for any man's ears may not be more than two or three years. Exposure to the average industrial noise for two or three years usually produces only minor changes in hearing.

These statements are based on the result of our experience with continuous noises. We know very little about the auditory effects of impact noises such as blasts, rifle fire, or drop hammer sounds.

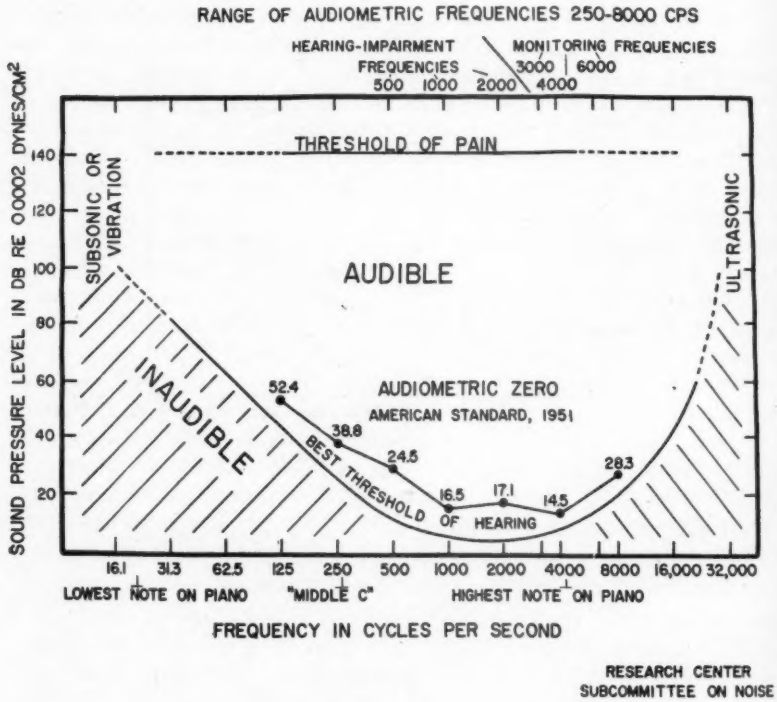

FIG. 7.

Psycho-Acoustics

In general, the young human ear can hear between 20 and 20,000 cps. This is usually called the audible spectrum (Fig. 7). Sounds that are lower in pitch are called subsonic or vibration and those that are higher are referred to as ultrasonic. Although the ear can distinguish frequencies between 20 and 20,000 cps it is considerably more sensitive between 1,000 and 4,000 cps. Advances in age produce an increasing loss which starts at the high frequency end of the spectrum. After sixty, few persons can hear beyond 10,000 or 12,000 cps.

Some areas of the audible spectrum are more important than others. For example, the speech range as represented in a conventional pure tone audiogram, 500, 1,000 and 2,000 cps. The value at these frequencies will indicate quite accurately how well a person can hear and understand speech.

Another area in the audible spectrum important to the person interested in noise extends through 3,000, 4,000 and 6,000 cps. Although these frequencies have no significant relation to the understanding of speech, they are a sensitive indicator of the onset and progress of noise-induced hearing loss. Hearing tests are designed to test hearing by way of the external ear, called air conduction tests, and the skull, or bone conduction tests. We are primarily concerned with air conduction tests.

Before we discuss hearing loss, let us first decide what constitutes normal hearing! "Normal hearing" is defined as the average hearing of a group of persons considered essentially representative of the population. Normal hearing has been designated only for the following frequencies: 64, 125, 250, 500, 1,000, 2,000, 4,000, 8,000, and 12,000 in the audible spectrum. The three frequencies, 64, 125, and 12,000, are not now used because they have been found to be unreliable as well as unnecessary. For any one of the chosen frequencies hearing is measured in units called "decibels of hearing loss".

Remember that the reference points for normal hearing are average values obtained by testing a large group of individuals. An average figure implies that many of the individuals tested are either better or worse than average. Because of this, normal hearing should not be thought of as a specific level, but rather as an area extending 15db. on either side of the average value.

Generally, hearing tests are given for one or more of three reasons: (1) as an aid in diagnosis, (2) for determination of amount of impairment, and (3) to monitor hearing conservation programmes. Diagnostic testing requires precise methods that will record the status of the external, the middle, and the inner ear at specific points of the audible spectrum. Diagnostic testing is an extremely important operation and should not be performed by other than medically approved audiologists. Only expertly produced data can afford the information necessary to diagnose and treat hearing loss.

There are three reasons for measuring hearing in industry, also: (1) for compensation purposes, (2) job classification prior to pre-employment, and (3) hearing conservation. Pre-employment audiograms must be made part of any routine physical examination given to employees prior to hiring. Further, because loss of hearing has become such an important problem to industry, pre-employment audiograms should become a routine procedure even if there is no routine medical physical examination. Pre-employment or pre-placement audiograms serve two

distinct purposes. The first and probably the most important is to establish a baseline hearing status prior to beginning work. The importance of establishing this baseline cannot be over-emphasized. An examination of audiograms made prior to hiring has shown that in most industries at least twenty-five per cent. of the persons examined have a hearing loss which may prove significant in case of a compensation claim later on. One of the most difficult problems in settling claims for hearing loss is establishing the status of the hearing in the applicant at the time of initial employment by the employer against whom the claim has been filed. Without pre-employment audiograms it becomes nearly impossible to determine just how much hearing loss has been sustained while working for the employer against whom the claim has been made, as well as being extremely difficult to assign responsibility. Frequently, the last employer is held responsible for the entire hearing loss unless he is able to show by a pre-employment audiogram that employment by him has produced only a small part of the total loss or perhaps no loss at all. Because the audiograms in noise-induced hearing loss cases and those with many other types of hearing loss that occur in the average worker are very similar, it is impossible to differentiate between them except when careful pre-employment audiograms and pre-employment otological histories are available. Without pre-employment audiograms, the status of the hearing prior to employment is merely a matter of opinion. Even if the workman is honest in his appraisal of his own hearing, he may have had a loss of which he was unaware.

In my opinion, pre-employment audiograms should not be used as a means of discriminating against employees at the time of hiring. Pre-employment audiograms can and should be used as a means of classifying employees according to the need for their hearing. What are these needs? How severe must a hearing loss be before it causes difficulty? (Fig. 8).

FIG. 8.

HEARING LOSS CLASSIFICATION TABLE.

AVERAGE DB LOSS AT 500, 1,000, 2,000 C.P.S. IN BETTER EAR.*

Class	Name		Remarks
I	Normal	Less than 15	Within Normal Limits.
II	Near normal.	More than 15 Less than 25	No difficulty with ordinary conversation at distances up to 20 feet.
III	Mild Loss.	More than 25. Less than 40.	Difficulty with ordinary conversation when distance exceeds 5 feet.
IV	Moderate Loss.	More than 40. Less than 65.	Difficulty with loud conversation when distance exceeds 5 feet.
V	Severe Loss.	More than 65. Less than 75	Difficulty with shout when distance exceeds 5 feet.
VI	Profound Loss.	More than 75. Less than 85.	Difficulty with shout at less than 5 feet.
VII	Practically total Loss	More than 85.	Loss of practical hearing for speech.

* This scale refers solely to hearing and does not take into consideration a man's competence with hearing aids, lip reading, (speech reading), etc.

The Effects of Noise on Hearing

How can we measure impairment due to hearing loss? Before a scale of handicap can be formulated, it must relate to an aspect of hearing loss that can be used as a common denominator. If we accept hearing and understanding speech as the most important function of the ear, a measure of handicap through hearing loss must be based on the effect of hearing loss on communication by speech. This effect can be quite accurately estimated by averaging the losses at 500, 1,000, and 2,000 cycles per second or at the so-called "speech frequencies".*

A Scale of difficulty due to hearing loss has certain points that are important. These points may be designated by averaging the values in db. at the speech frequencies of 500, 1,000, and 2,000 cps. The first important point on a hearing loss scale is 15 db. It marks the outside limit of normal hearing. In other words, a person who needs greater volume than this to hear may be said to have impaired hearing. When the loss increases to between 20 and 30 db., most persons notice a slight difficulty under certain circumstances. When the loss progresses beyond 30 db., the difficulty increases noticeably. Hearing loss at this level becomes evident in the day-to-day activities of the afflicted person. As the hearing loss progresses, a third stage is reached at 45 db. where one notices considerable difficulty with communication under most circumstances.

The next level of consequence is at 60 or 65 db. With losses of this magnitude a person cannot communicate without serious difficulties. The final point on this scale is 80 or 85 db. A hearing loss of this magnitude totally precludes any practical speech communication. Naturally, the ear will respond to sounds that are louder than this but the human voice is not capable of continuous speech at this level for more than very short periods.

Pre-employment hearing tests are perhaps the most important procedure in a hearing conservation programme. These tests will act as a baseline with which to compare follow-up hearing tests. Follow-up tests when routinely given to persons who work in excessive noise levels will act to monitor the effectiveness of the hearing conservation programme. Further, they will serve to discover the highly susceptible persons who may be hired.

Pre-employment or pre-placement audiograms should be air conduction threshold tests. The term air conduction describes the path by which the test sounds reach the ear. In air conduction tests, the test tones are produced by ear phones and conducted through the air in the ear canal to the drumhead. Recommended test frequencies are 500, 1,000, 2,000, 3,000, 4,000, and 6,000 cps. The record of measured thresholds is called a "threshold air conduction audiogram".

Pre-placement threshold audiograms should be made for *all* employees,

* *Guide for the Evaluation of Hearing Impairment*, Transactions of the American Academy of Ophthalmology and Otolaryngology, March-April, 1959.

not only for those who are to work in noisy areas. Such a record may show, for example, the presence of early hearing loss of a type that is responsive to suitable therapy. Detection and treatment of these non-noise-induced hearing losses will help prevent their subsequent progress and the resulting loss of trained personnel.

If a pre-placement audiogram shows an average hearing loss of more than 15 db. at 500, 1,000 and 2,000 cps, the employee should be referred to a physician for otological examination and placement evaluation before assignment to a job. Such referral is also advised for employees whose audiograms show any unusual irregularity, particularly an abrupt loss beginning at 2,000 cps.

Whenever pre-employment and/or pre-placement tests are given, the tester should ascertain that a period of at least sixteen hours has elapsed since the subject's last noise-exposure.

Test environment

Acoustical treatment of audiometry rooms may prove to be a difficult task and should be undertaken only with the help of an acoustical consultant. Pre-fabricated audiometry booths usually are more satisfactory and may be cheaper than either reconstructed rooms or sound treated rooms (Fig. 9).

Recording the Data. A specially designed hearing conservation data card and instructions for its use may be obtained from the Research Centre of the Sub-committee on Noise. The card is designed so that its contents can be transferred quickly to punched cards, thus facilitating the rapid analysis of large amounts of data (Fig. 10). The card need not be filled in completely but users are encouraged to check as many of the items as possible. A completed card contains the minimum amount of information needed to make a research study of the relations of hearing loss to noise-exposure. An experienced tester requires no more than six minutes to complete the hearing test and about three minutes to fill in the card.

Recent Research

A study of various populations is reflected in Figs. 11 and 12. These median audiograms (1,000 and 4,000 cycles per second) are based on large groups that are representative of the populations indicated. It is important to note that the general population taken from two large Fair surveys and the typical industry sample are very much the same, but show a significant difference when compared with a large group selected on the basis of minimum noise exposure. Of particular importance is the marked elevation of the threshold at these two frequencies in the group designated as 105 db. overall.

The typical industry group is exposed to over levels varying from

FIG. 9.

HEARING CONSERVATION DATA CARD NO. _____ TYPE OF AUDIOGRAM REFERENCE AND/OR PRE-EMPLOYMENT ☐ RECHECK ☐ OTHER _____

A. IDENTIFICATION

LAST NAME	FIRST	MIDDLE	SEX	DATE OF BIRTH		
			MALE FEMALE	DAY	MONTH	YEAR
SOCIAL SECURITY NUMBER			COMPANY NUMBER			

B. CURRENT NOISE EXPOSURE

JOB TITLE OR NUMBER*	TIME IN JOB	NOISE EXPOSURE*		% TIME NOISE ON	EMPLOYEE ESTIMATE OF OWN HEARING *
	HOME _____	STEADY NOISE	IMPULSE NOISE		GOOD
DEPARTMENT OR LOCATION*	MOS. _____	CONTINUOUS ☐	CONTINUOUS ☐	10 20 30 40 50	FAIR
	YRS. _____	INTERMITTENT ☐	INTERMITTENT ☐	60 70 80 90 100	POOR

C. AUDIOGRAM

AGE	TIME SINCE MOST RECENT NOISE EXPOSURE*					DURATION OF MOST RECENT NOISE EXPOSURE*			EAR PROTECTION	
	0-20 MIN	1 HR	4-7 HRS	1 DA	4+ DAS	0-20 MIN	1 HR	4-7 HRS	WAS EAR PROTECTION WORN?	YES
	21-50 MIN	2-3 HRS	8-16 HRS	2-3 DAS		21-50 MIN	2-3 HRS	7+ HRS		NO

DATE OF AUDIO	DAY OF WEEK	TIME OF DAY	RIGHT EAR								LEFT EAR							
			250	500	1000	1500	2000	3000	4000	6000	250	500	1000	1500	2000	3000	4000	6000

D. PREVIOUS NOISE EXPOSURE & MEDICAL HISTORY*

PREVIOUS EMPLOYMENT (LAST 3 JOBS)*

TYPE OF WORK	FOR WHOM	HOW LONG

HISTORY			RECORD ANY COMMENTS SUBJECT MAKES ABOUT HEARING*
HEAD INJURY (WITH UNCONSCIOUSNESS)	☐		
HEARING LOSS IN FAMILY (BEFORE AGE 50)	☐		
TINNITUS FOLLOWING NOISE EXPOSURE	R	L	
STATUS			
PERFORATIONS OF EARDRUM	R	L	
DRAINAGE FROM EAR	R	L	
MALFORMATION OF EAR	R	L	

TECHNICIAN _____ PHYSICIAN _____

RESEARCH CENTER SUBCOMMITTEE ON NOISE. NCDC 1958 M. REVISED JUNE 1958

FIG. 10.

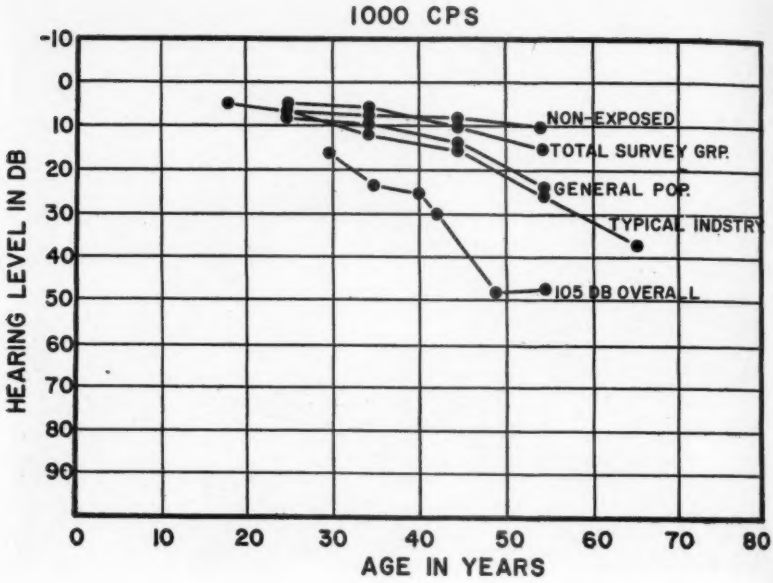
1000 CPS
HEARING LEVEL IN DB
-10
0
10
20
30
40
50
60
70
80
90
0
10
20
30
40
50
60
70
80
AGE IN YEARS
NON-EXPOSED
TOTAL SURVEY GRP.
GENERAL POP.
TYPICAL INDSTRY
105 DB OVERALL

FIG. 11.

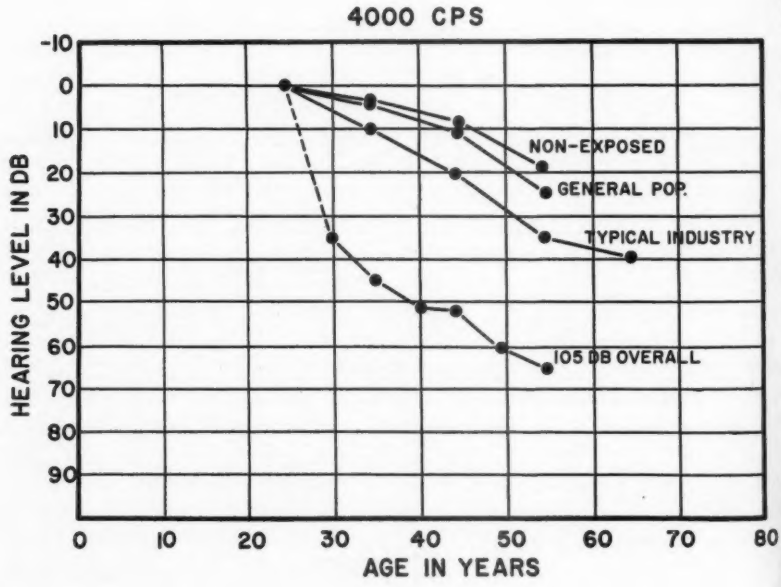
4000 CPS
HEARING LEVEL IN DB
-10
0
10
20
30
40
50
60
70
80
90
0
10
20
30
40
50
60
70
80
AGE IN YEARS
NON-EXPOSED
GENERAL POP.
TYPICAL INDUSTRY
105 DB OVERALL

FIG. 12.

95 db. to 115 db. overall but the time distribution is varied by intermittency of exposure and relatively rapid job turnover. Whereas the group designated as 105 db. overall is exposed continuously for more than five hours per day for many years. We have called the former a heterogeneous exposure as opposed to a homogeneous exposure in the latter case.

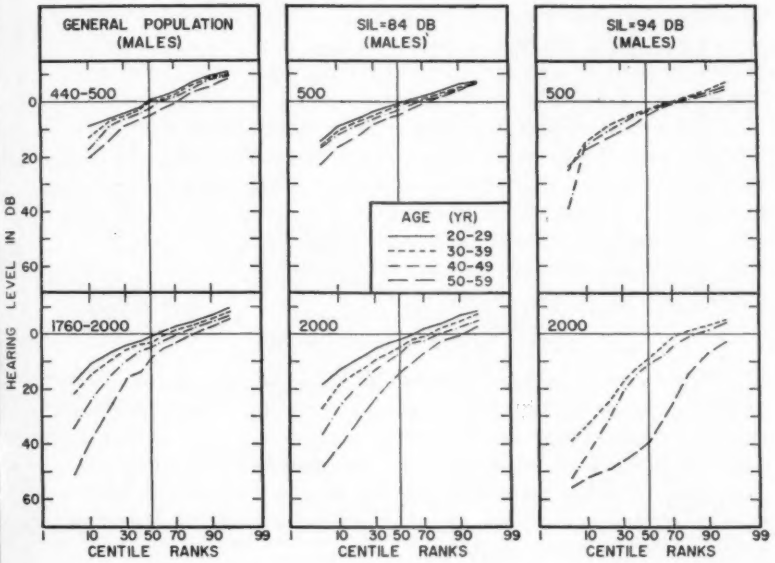

FIG. 13.

We believe that intermittent exposures are far less damaging to the ear than continuous exposure even if the sound pressure levels are considerably higher. The fact that the ear has a chance to rest for short periods seems to be very important.

Now look at Figs. 13 and 14. These show a comparison of three populations. The data are distributed according to centiles, showing the percentage of males with specific losses at 500, 1,000, 2,000, and 4,000 cps. The samples contain males taken from a general population survey (20, 459 men), males taken from an industrial group that has been exposed to 84 db. at 600-1,200, 1,200-2,400, and 2,400-4,800 (SIL bands) continuously for more than five hours per day (9,653 men) and 338 males who have been exposed to 94 db. in the same octave bands continuously for more than five hours per day. Note the general similarity in distribution by centiles between the general male population and the SIL 84 db.

population. Note that the similarity holds throughout all frequencies except 4,000 cps Now compare the SIL 94 db. group with the other two. The difference is striking at all frequencies except 500 cps and even at this frequency there appears to be more hearing loss.

Fig. 15 presents a composite of the analysis of a large amount of data. The curves present median hearing level at 4,000 cps as a function of continous exposure (five hours or more per day) in years and as a function of the average group age relative to exposure years. Exposure is to 1,200-2,400, and 2,400-4,800 cps. Our experiments show that there is only a slight difference in the effect on hearing at 4,000 cps between these two bands.

Notice two important points in these data. When the exposure level is approximately 80 db. in these octave bands the change in threshold is very little different than that produced by ageing alone. Second, when the exposure level is high enough to produce a threshold shift, the maximum shift for each exposure condition occurs in approximately 10 years. Any subsequent change in threshold appears to be strictly a function of ageing.

Damage Risk Criteria

With these thoughts in mind, let us consider damage risk criteria a moment. The search for damage risk levels is certainly not confined to noise-exposure. There are exposures such as noxious air contaminants, solvents producing skin irritation, radiation hazards, ultraviolet burns, and many others for which setting damage risk levels has been, and still is, a major problem. Firm damage risk levels have not been established for many industrial hazards.

The history of attempts to control these hazards is filled with illustrations of the difficulties associated with establishing damage risk levels for any industrial hazard whose effects are measured by changes in biological structures. Most potentially dangerous industrial exposures can be measured by physical or chemical methods; but the important measure of a hazard of this nature is its effect on the exposed person. Measuring the biological effects of hazardous industrial exposures is seldom a simple matter. For example, radiation from fissionable products can be measured quite accurately but the biological effects of radiation are difficult even to estimate until gross changes occur. On the other hand, lead poisoning can be accurately followed by a simple blood test.

In general, a damage risk level can be defined as a point or series of points on a scale measuring as a function of exposure the hazardous bio-effects of a chemical or physical agent. The scale extends between the limits of no pathology and fatality. The damage risk level designates the maximum allowable levels for which no significant untoward bio-effects will occur within a specified exposure period.

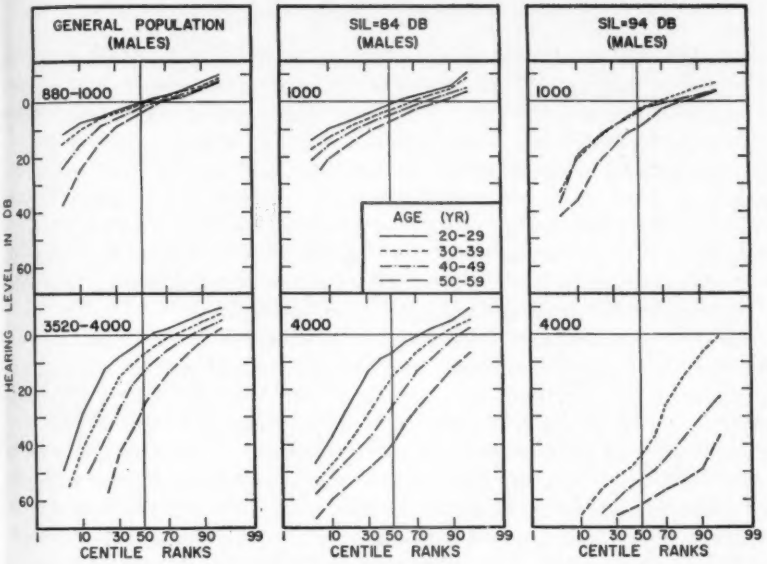
GENERAL POPULATION (MALES)
SIL=84 DB (MALES)
SIL=94 DB (MALES)
880-1000
1000
1000
3520-4000
4000
4000
HEARING LEVEL IN DB
AGE (YR)
20-29
30-39
40-49
50-59
CENTILE RANKS
CENTILE RANKS
CENTILE RANKS

FIG. 14.

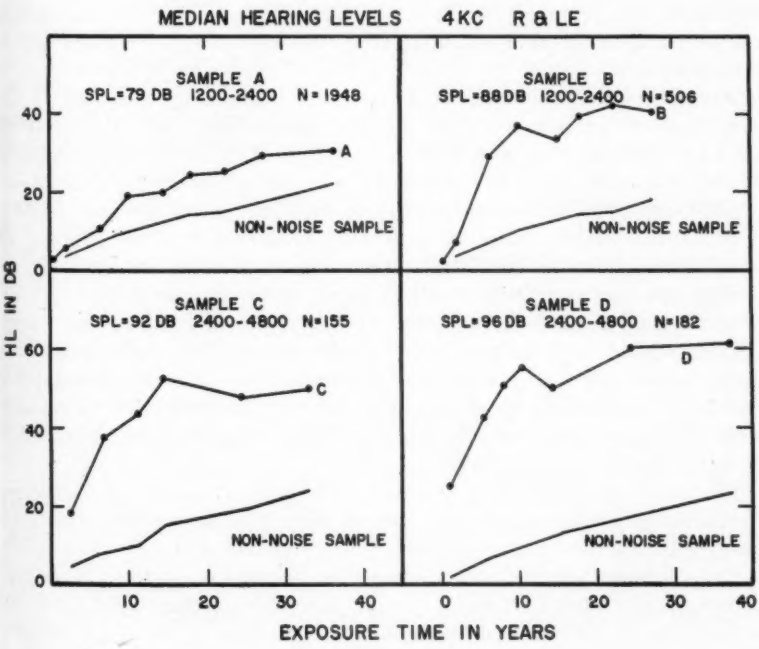
MEDIAN HEARING LEVELS 4 KC R & LE
SAMPLE A
SPL=79 DB 1200-2400 N=1948
SAMPLE B
SPL=88 DB 1200-2400 N=506
SAMPLE C
SPL=92 DB 2400-4800 N=155
SAMPLE D
SPL=96 DB 2400-4800 N=182
NON-NOISE SAMPLE
HL IN DB
EXPOSURE TIME IN YEARS

FIG. 15.

Noise-exposure. We are concerned today with the specific problem of noise and its damage risk level. Our chief concern in the early consideration of this problem was to set damage risk noise levels. Experience with other industrial hazards and an increase in our knowledge of the relations of hearing loss to noise have emphasized the fact that we must deal not with noise levels but rather with noise-exposure limits. Obviously, noise without exposure is no hazard. Noise is defined as unwanted sound. The definition of noise-exposure is more complex. Noise-exposure must be defined in terms of the overall level and character of the noise as a function of the time distribution and total duration of exposure.

When the concept of *noise-exposure* is accepted it becomes obvious that industries vary considerably as to the amount of *risk*. Some industrial operations have very high noise levels but require only short exposures, either in terms of interrupted periods of exposure throughout the day and/or a short total duration of exposure. Similarly, some plants may have a very high noise-exposure both in level and time but only a few exposed personnel. Conversely, other plants may have a relatively low noise-exposure with many exposed personnel. In the latter case, even though the noise levels may be distinctly lower, the risk is much higher. Surveys of the industry in general show that few manufacturing plants are without noise-exposure problems, indicating that noise-exposure is widespread and demands immediate and concerted attention if its bio-effects are to be counteracted.

Limitations to Damage Risk Criteria. Setting damage risk levels for noise-exposure presumes a great deal of knowledge about many related factors. Before any designated limits for noise-exposure can be established we must, for practicality's sake, assume certain compromising limitations. No noise-exposure limit can be set to protect everyone. This is not possible, let alone reasonable; there is individual variation in susceptibility to noise-induced hearing loss. The effects of susceptibility vary from total hearing loss in one ear as a result of exposure to one pistol shot to very minor hearing losses from exposure to repeated gun fire over very long periods. We are not prepared to explain susceptibility nor to predict who is more highly susceptible and therefore, we cannot expect to establish criteria for preventing hearing loss in anyone and everyone. We must be satisfied to set practical criteria based on the laws of probability which will protect as many persons as possible.

We must determine such practical criteria on the basis of industrial life-time exposure to noises with various time distributions as well as to noises with various energy distributions. Criteria will vary depending on the character of the noise; steady or impact, continuous or intermittent. Further, we must accept the fact that it is not practical to prevent hearing loss in the entire audible range. With this in mind it becomes important to establish areas of relative importance in the audible spectrum

usually measured by the audiometer. If we accept hearing and understanding speech as the most important single function of the ear, and I believe we must, it is obvious that prevention of hearing loss in the speech frequencies is the least we should expect from any damage risk criteria. Ideally, of course, our criteria should prevent any hearing loss but practically this is frequently not possible.

If we carry this reasoning to its logical end, it is obvious that the ultimate purpose of our criteria should be to prevent a social and/or an economic impairment to hearing loss. If we accept such an idea, and again I believe we must, we are directed to the hearing and understanding of speech as the most acceptable common denominator upon which to rate handicap due to hearing loss and as the ultimate basis for establishing damage risk prevention criteria.

Hearing Conservation Criteria. It is quite evident that we must learn a great deal more about the relations of hearing loss to noise-exposure before damage risk criteria can be established or, for that matter, even suggested with any degree of validity. What then must we do in the meantime?

In the absence of enough information to fix criteria, we must resort to noise-exposure limits related to hearing conservation. We cannot propose "safe" levels, only conservative limits that indicate when it is advisable to initiate a hearing conservation programme.

These "limits" cannot be general because of the many different types of noise and different schedules of exposure. The hearing conservation limit that we now specify tentatively applies only to years of exposure to broad band steady noises with relatively flat spectra. It does not apply to short exposure and, above all, it does not apply to impact noises or narrow band noises. This tentative hearing conservation limit is stated as follows:

> If the sound energy of the noise is distributed more or less evenly throughout the eight octave bands, and if a person is to be exposed to this noise regularly for many hours a day, five days a week for many years, then: if the noise level in the 600 to 1,200 cycle band, the 1,200-2,400 cycle band, is 85 db., the initiation of noise-exposure control and tests of hearing is advisable. The more the octave band level exceeds 85 db. the more urgent is the need for hearing conservation.

The 85 db. is *not an overall sound pressure* level; it refers to two particular octave bands. The overall level of the noise is always higher than the level in any one octave band, at times as much as 20 db. higher.

It is quite clear then, that we must be content with hearing conservation limits until we can affix more realistic damage risk criteria.*

It is quite apparent that when the exposure is continuous and above

* *Guide for Conservation of Hearing in Noise*, Noise Control, Vol. 3, No. 3, May, 1957.

85 db. in the octave bands given above hearing loss will result. It is apparent also that most of the loss occurs in the higher frequencies (above 2,000 c.p.s.). However, note that even the frequencies important to communication by speech (500, 1,000, and 2,000 c.p.s.) are shifted if the exposure continuous for 10 or more years when the band level is 95 db. or above. With such conclusive evidence it behoves industry to initiate hearing conservation programmes, including (1) pre-employment, preplacement and periodic audiograms; (2) noise reduction and/or personal protection programmes. The long term benefits of a hearing conservation programme cannot be overestimated.

I am convinced that the potential cost of noise-induced hearing loss to industry is greater than any other occupational disease. I am, also, convinced that if a common sense attitude on the part of management and labour is not adopted the precedents that may be set will lead to serious problems. On the other hand, I am sure that with co-operative effort by all concerned, not only will the potential cost in dollars be reduced to a livable minimum but noise-induced loss of hearing can be reduced to a negligible amount.

Admittedly, the solution calls for understanding, patience, and an enormous amount of education. Further, I do not expect it will be settled in a few months. On the contrary, I am sure it will take quite a few years plus a lot of intensive research. But if we can preserve hearing in the large majority of industrial employees, it will be worth our efforts.

327 S. Alvarado Street,
Los Angeles 57,
California, U.S.A.

SOME OCCUPATIONAL EFFECTS OF NOISE

By DONALD STEWART (Birmingham)

WITHIN British industry, with one or two notable exceptions, little significant research has been carried out on the occupational effects of noise on health or on human performance. The following introduction to one of these exceptions (Pollock and Bartlett, 1932), although written nearly thirty years ago, still holds good today.

> "The effects of intense and continuous noise on the human mind and body have been far more frequently a matter of popular comment than of scientific study. There appears to be a widespread opinion that noise directly diminishes efficiency, leads to increased liability to accidents, and provides a persistently favourable background for the development of nervous and mental disorders of many kinds. For all of these opinions there is very little evidence beyond the fact that noise tends to be disliked, particularly by people who are not very much accustomed to it."

In industry the problem of noise can be considered from several points of view: the effect on hearing and health, the effect on human performance, hearing conservation programmes, and methods of reducing noise.

Effects on Hearing and Health

Boilermakers' deafness was first described early in the nineteenth century, blacksmiths' deafness in 1831, and cotton-weavers' deafness in 1925. These are well-known classical examples of occupational deafness. But it was not until 1953 that further field studies were described about the auditory effects of noise exposure, not only in boilermakers but also in other factory groups such as drop-forgers and machine operators in the screw manufacturing industry (Johnson, 1953).

This survey was sponsored by the Medical Research Council under the general supervision of Terence Cawthorne, and I had the privilege of providing the field for investigation in factories where I acted as medical adviser. I was thus fortunate in having an opportunity to learn something about the effects of noise at first-hand during frequent discussions with the investigators.

Johnson showed that among 219 men and women working in selected occupations known to be noisy, such as boilermaking and drop-forging, 28 suffered from bilateral ear disease of infective or degenerative origin

not due to acoustic trauma; and of the remaining 191 cases all but one were found to have hearing losses due to noise which could be demonstrated either by speech or pure-tone audiometric tests. Noise measurements suggested that only when the overall noise level exceeded 105-108 phons is serious damage likely to be caused to hearing. This is the generally accepted view of other observers. At this level the ear must be protected by some suitable ear defender.

Personal impressions remain with me resulting from discussion both during and after this survey, with a number of the men concerned. With few exceptions boilermakers and drop-forge stampers were aware that deafness occurred in their industry. But this caused no worry nor was it considered in any way serious, it was just accepted as part of the job. The insidious course of their deafness had made them unaware of the degree of hearing loss. Many of the older men still had serviceable hearing, and some had learned to lip-read. Social experiences were interesting. Complaints of trouble or worry at home because of deafness were rare. Men were perfectly willing for their sons to enter their trade. Deafness was perhaps an excuse to avoid union meetings or going to church. Television had not yet "arrived", but the cinema was well patronized, more often for vision than the spoken word and a number confessed to lip-reading. Several men in the younger age groups with less exposure said that hearing improved after leaving work, particularly at weekends and during holidays.

During some 15 years the number of complaints of deafness which came to my notice among boilermakers and stampers was small. The men frequently asked that wax should be removed to make them hear better, and it did. Many of them had this done every 6-12 months. A few preferred to retain their wax, for they felt it was a form of protection. Ear defenders although available were often discarded. Health complaints for other reasons were seldom linked with loss of hearing. Only on the rarest occasion was it held to be a cause of accident.

Effects on Human Performance

In 1932 Weston and Adams published a report of controlled studies on the performance of operatives in a noisy Lancashire weaving shed. Performance was measured by recording the output of each loom. Where loom noise was reduced by using ear defenders the hourly average output per weaver in the group studied showed an increase of approximately one per cent. They therefore claimed to have established that noise could interfere with industrial output in this particular industry. They also showed that even after years of work in a noisy environment employees do not become completely adapted or acclimatized to noise but go on daily through the process of adaptation. During initial working periods when adaptation is minimal the average loss of output may be as high as 3 per cent. in individual cases.

The experiment, the result of which has not so far been disproved, underlines the need for further work on the same subject. It seems probable that in other occupations with comparable noise intensities, but depending less on mechanical factors and more on the human factor, the effect of noise on output might be considerably greater than that demonstrated by this investigation.

The measurement of human performance provides many difficulties the most important of which is the unreliability of subjective assessments. Factors affecting performance are: labour turnover (that is the percentage of the total factory strength leaving each year); absence from work due to sickness, accident, or without leave; personal likes and dislikes caused by lack of satisfaction, by emotional factors, or by inadequate leadership; hours of work and type of work; and physical working conditions such as lighting, heating, ventilation, fumes or dust, and noise, all resulting in fatigue.

It can be seen therefore that the isolation of noise as the main cause or even as one cause of decreased output or of ill-health, is not by any means easy. Further research within this field is essential particularly in less noisy workplaces, and could come within the sphere of operational research now developing rapidly in various industries, assisted in the first place by research groups outside industry.

Present-day Developments

There is evidence of new activity on noise problems in Government departments and at research centres. Perhaps noise is no longer to be accepted as an inevitable part of modern living. A Noise Abatement Bill received its second reading in the House of Commons on the 4th March, 1960. Although this deals particularly with the noise of road traffic it is perhaps relevant to this discussion. It is indicative of a public reaction which, through the Press, now demands action in the whole field of noise. And it highlights a major difficulty in promoting legislation to deal with the problem, namely the lack of an accurate method of measuring noise.

It is perhaps significant that the technical officers of the Ministry of Transport are said to be collaborating with the International Standards Organization to establish practical methods of estimating noise. But before coming to any conclusion the Ministry will no doubt seek the advice of various research bodies in this country, in particular the Department of Scientific and Industrial Research and the Medical Research Council.

An article on the problem of noise in industry (*Times*, London, April 1st, 1960) says that pocket-size sound-level noise-meters have recently been produced. These have been used in banks for noise surveys

with apparent success as a first step to introducing methods of reducing the noise created by mechanization.

As an aside to the industrial problem it is interesting to know that the Southend Police have been using similar instruments to measure noise levels in evidence to support prosecutions of motorists and transport drivers. As another aside a recent report (*Daily Telegraph*, London, April 26th, 1960), said that the Italian manufacturers of the Vespa motorscooter had offered to make all their scooters now in use on Italian roads less noisy by fitting efficient silencers free of charge. But British dealers were opposed to this on the grounds that sales would drop, for many owners liked the noise.

It was reported recently (*Lancet*, April 23rd, 1960) that the Government had set up a committee "to consider noise and its effects". The committee includes two doctors, one a psychiatrist and the other a medical officer of health, but no otologist.

Should Occupational Deafness be Classified as an Industrial Disease?

This is primarily a question for the Government through the Ministry of Pensions and National Insurance. One difficulty is the identification of the cause as between the normal ageing process and specific occupations. A recent issue confronting otologists, and relevant to the question, is that claims may arise for monetary compensation because of potential loss of earnings where they advise job changes. Deafness is classified as an industrial disease in France, Germany, Finland and in Russia, but so far not in Great Britain. Under British legislation if occupational deafness is to be a "prescribed" disease, so also has the occupation to be "prescribed". Only thus can claims be made for loss of physical and mental faculty. And here the real difficulty arises. Occupational deafness would have to be treated as *a risk of occupation* and not as a risk common to all persons. Attribution of particular cases to the nature of the employment would have to be established. This is a matter which no doubt will be reviewed by the Industrial Injuries Advisory Council of the Ministry of Pensions and National Insurance on which medical experts serve as members as well as representatives of the trades unions and employers' associations.

Medical Supervision at Work

It has from time to time been suggested that what our American friends describe as a "hearing conservation programme" should be put into effect in noisy trades, and that this must be the final responsibility of the industrial medical officer. But before noise can be held to be hazardous in any occupational group it is essential that a survey of the noise conditions should be carried out. By whom this could be done, and from what source could the experts be drawn, are questions I would like to put

to you. For example, could this come within the province of some new occupational hygiene laboratory service? In this country we have much to learn from American states where such laboratories are well-established and give a much needed service to local factories and business organizations.

A hearing conservation programme is held to include pre-employment aural history and ear examination, including pure-tone audiometry. Periodical re-examination would be essential. But again by whom could this best be done? By the industrial doctor alone, or with technical assistance, or under the guidance of a consulting otologist with a special interest in preventive medicine?

Are otologists ready or willing to enter this field? In addition to hospital and practice commitments would they be able to give time to initiate and supervise special training schemes for industrial medical officers? On one point let us be quite clear: without this training no hearing conservation programme in industry could be fully effective.

Noise Suppression

Extreme noise is relatively uncommon in industrial concerns. Where it occurs management in my experience appears to be anxious to take steps to reduce it, but not by spending large sums of money. Although deafness can probably be accepted as an occupational hazard in occupations such as boilermaking or aircraft engine testing, the degree of hazard has yet to be established in many other trades before employers can reasonably be advised to carry out extensive alterations to their plant and processes.

A noise suppression programme would basically include attempts at silencing, isolation, and sound-proofing, as well as the introduction of methods of ear protection. This immediately creates an interesting issue for the matter becomes a joint enterprise between employees, managers, engineers, and doctors. How often could we expect to find a suitably trained and experienced person within any industrial concern able to advise on this? Where could we find an individual outside industry able to act as a consultant in such matters? Would top management co-operate, particularly when it came to cost, for cost is an important obstacle to noise suppression? To what extent do architects and construction engineers consider the prevention of noise in their building projects? Are they fully aware of their responsibility in the matter? In their training is sufficient emphasis placed on the prevention of noise?

Conclusion

Little is known about the effects of noise on the health and efficiency of industrial workers apart from deafness. There is need therefore to implement the work of Johnson, and that of Weston and Adams, by

operational research within industry, and where possible by industry. Assistance in this field should become available from otologists and experts in industrial health research, with the co-ordinated assistance of social workers, industrial psychologists, engineers and architects. One primary objective of such investigations should be the preservation of hearing.

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2 Hagley Court,
Hagley Road,
Birmingham, 16

THE EFFECTS OF NOISE UPON HEARING

By E. D. D. DICKSON (London)

THERE is no doubt that with increasing mechanical power the intensity of traumatic agents is bound to make a greater impact on the ear.

Emphasis, however, especially in this country, has been and still is directed more towards the nuisance value of noise and not enough stress laid on its effects on Corti's organ. The psychological effects have been emphasized to the exclusion of the otological. The otologist must realize he has a vital role to play in any deliberations that have bearing on such matters as prevention of noise, conservation of hearing and compensation. And it is the importance of prevention which I would like to stress. The primary objective any conservation of hearing programme must have is the preservation of the ability to hear, and its secondary one that of protection against unjustified compensation claims.

I cannot stress sufficiently that the medical aspects of any such programme should be under the direct supervision of someone who by training and interest is well acquainted with the technical aspects of the problem. The cornerstone of any future assessment, whether for compensation purposes or continuation in a noisy occupation, is the otologist's evaluation of the extent and cause of any hearing loss, if present. This would constitute a more acceptable proof of loss if such data be needed at a later date. It will at any rate ensure that the employer knows at least the amount of hearing loss, even if it occurs five to ten years later, for which he is likely to be held responsible.

Once pre-exposure and periodical audiometric evaluation have been performed, hearing conservation becomes a real thing by protecting those individuals who demonstrate progressive noise-induced hearing losses and those who show undue susceptibility to noise.

A word about ear protection.

A good ear protector is a safe-guard for all but the most extreme noise exposure. Ear protection will not deprive the worker of hearing necessary for speech or warning signals. If success in any attempt to protect hearing is the aim of all concerned, then supervision becomes necessary. General guidance, distribution and fitting should be the responsibility of the individual medical officer, but responsibility to make certain that any protective device adopted is used must rest with the plant foreman. Only, and only if the men, the foreman and the management have appreciated the value of ear protection, can any scheme operate efficiently.

The actual amount of protection or attenuation possible to a specific individual is determined by the fit of the device, its physical condition and the willingness and ability of the individual to use it properly.

We must realize that industrial deafness is a fairly new concept which will have to be fitted into the whole pattern of workmen's compensation sooner or later.

Some important questions will have to be answered and defined, and I will put these forward for your consideration:

(a) What noise levels in industry are injurious?
(b) What is the proposed method of determining the employee's hearing loss resulting from his occupation as distinguished from other causes?
(c) What hearing losses are disabling and what criteria should be used in defining disability?
(d) How will we deal with the problem of accrued liability?
(e) What frequencies should be used in measuring industrial loss?
(f) Should monaural or binaural methods be used for computing percentage loss of hearing?
(g) What is the number of examinations needed to evaluate industrial hearing loss and the fairest method of determining the loss from the results of successive examinations?
(h) What is the point below which there is no hearing disability and the point above which the inability to hear shall be deemed total?

Lastly, we must consider the proper deduction for presbyacusis and other non-industrial causes of hearing impairment.

Enough has been said to emphasize the importance of prevention and protection.

Any formula adopted should be predicated on sound scientific principles because until we know the true degree of the occupational hearing loss and when it can be regarded as permanent it is difficult to determine the fair compensable value of the impariment.

There is no single magic number yet available which constitutes the dividing line between hazardous and non-hazardous exposure. Before the question of attributability is settled a six-months waiting period is generally advocated because it involves the principle of recovery from acoustical fatigue and thus defers awards until after a reasonable period of separation from the job.

Minor hearing losses should be excluded from compensation coverage because they neither cause social inconvenience nor are they outside the limits of normal variability.

Royal Air Force,
Kelvin House, Cleveland Street,
London, W.1.

PRE-EPIDERMOSIS

By A. TUMARKIN (Liverpool)

THE word Cholesteatoma is deeply embedded in Otological literature and is generally regarded as signifying a definite clinical entity. Unfortunately that is not strictly correct. Not only is there controversy as to its ætiology but there is ambiguity with regard to its histological characteristics.

Two main points call for discussion:

(a) Is it possible to demonstrate true stratified epithelium?

(b) Is the presence of cholesterin crystals an essential feature?

With regard to (a) informed opinion today is unanimous that stratified squamous epithelium *can be* demonstrated. There is, however, great controversy not only as to the *origin* of this stratified squamous epithelium, but also the means whereby it enters the middle ear.

As to (b) it cannot be too strongly emphasized that Cholesterin has nothing whatsoever to do with Cholesteatoma. Fig. 1 shows the classical picture of Cholesteatoma—the amorphous debris, the keratinized layers and the stratified prickle cells. Fig. 2 shows an entirely different entity. The outlines of the Cholesterin crystals stand out clearly and in addition giant cells, granulation tissue and blood pigments can be seen.

These are entirely different histological entities, and yet the word Cholesteatoma is regularly used to denote either or both of them. Friedmann 1959, has drawn attention to this confusion and has proposed to clarify matters by denoting the first "Epidermoid Cholesteatoma" and the second Cholesterol Granuloma.

The term "Cholesterol Granuloma" is scientifically accurate and highly evocative. It denotes a condition characterized by cholesterin, granulation tissue and giant cells. The term "Epidermoid Cholesteatoma" is, however, open to criticism since the word "Cholesteatoma" carries with it all the vague suggestions of Cholesterin and of neoplasm which we are anxious to eliminate. In the writer's opinion the word "Epidermosis" is preferable denoting as it does a "process" rather than a preformed condition.

Definition. Epidermosis of the tympano-mastoid cavity is a pathological process in which squamous epithelium of *epidermic origin* grows into that cavity.

By this definition we exclude two other theories of origin, viz.:

(1) The epiblastic theory which postulates congenital inclusions of epiblast, i.e. dermoid (or epidermoid) tumours. That such congenital

inclusions are possible cannot be denied but they are rare curiosities. In most cases, Epidermosis develops post-partum by an invasion of epidermis.

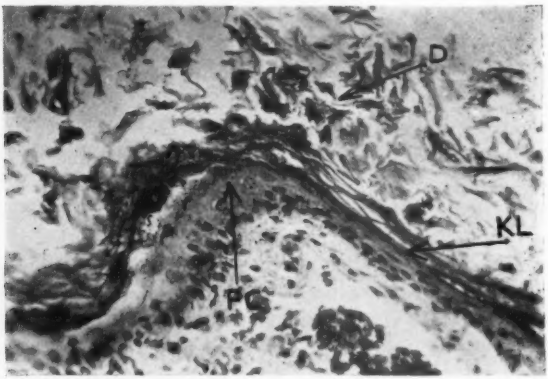


FIG. 1.
EPIDERMOSIS.
Showing prickle cells (PC), Keratinizing layers (KL), Debris (D).

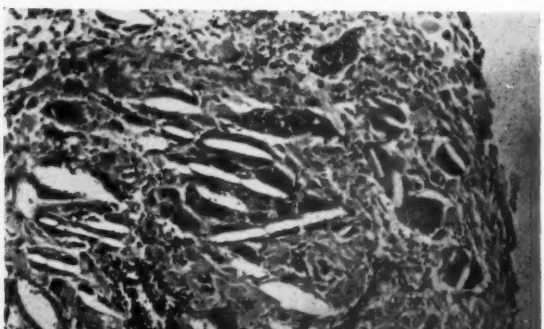


FIG. 2.
CHOLESTEROL GRANULOMA.
Showing typical clefts, giant cells, pigments and granulation tissue.

(2) The metaplasia theory. This in effect says that the stratified squamous epithelium is *not epidermic*, being derived from a transformation of the endothelial lining of the middle ear cleft.

Here again it must be conceded that metaplasia is a theoretical possibility.

Pre-Epidermosis

The normal mastoid is lined by a single layer of flattened cells. In acute mastoiditis these change into ciliated columnar cells. This itself is metaplasia and there is no reason to deny that a further transformation from ciliated columnar into squamous epithelium might occur just as it does in the nose, the bronchi and elsewhere. The possibility of this sort of two stage Metaplasia has been supported in one form or another by many workers (including the writer) ever since it was originally proposed by Wendt, nevertheless, no unequivocal evidence of its existence has so far been adduced and it is now generally agreed that the stratified squamous epithelium is derived from the skin of the meatus and tympanic membrane by a process of immigration.

The concept of immigration is, of course, one of the classical theories but on closer analysis it turns out to be far from simple. There are at least five different ways in which immigration is believed to occur and the distinction between them is no mere academic exercise—it has profound practical implications.

Immigration Theories

(a) *Mechanical.* Seventy years ago Bezold (1891) (and later Wittmaack (1918)) suggested that as a result of chronic tubal catarrh the drum collapses and forms a deep blind sac in which products of desquamation tend to accumulate, finally invading the middle ear cleft.

(b) *Inflammatory.* About the same time Habermann (1888) suggested that an acute necrotising otitis media could destroy the drum right down to the annulus after which the meatal epidermis could invade the middle ear. Even in those days, however, it was recognized that this could not explain the more common case characterized by an insidious course and a small marginal perforation.

(c) *Tissue Tension.* During the last decade Eigler (1949) has drawn attention to the peculiar histological features of the skin of the deep meatus—not only a tendency to hyperkeratosis of the stratum corneum, but also a tendency of the basal cells to form solid cones which proliferate inwards. This "Wachstum potenz" theory has been taken up vigorously by many workers. Schroer (1956) claims that it is quite capable of producing a cholesteatoma in the absence of any inflammation whatsoever. Rüedi (1957) modifies this extreme attitude by suggesting that the invasion is more likely to occur if the epitympanal spaces are incompletely pneumatized and that it is usually triggered off by an acute otitis media in infancy. Both these authors point out that this proliferation of basal cells could theoretically produce Cholesteatoma *behind an intact tympanic membrane.* Rüedi, however, adds (*loc cit.* p. 301), "It is unfortunate we do not yet have histological specimens of acute otitis media in which proliferated cones of basal cells have penetrated the connective tissue and

formed a cholesteatoma. Histological proof of an inflammatory development of a cholesteatoma within an intact Shrapnell's membrane is therefore still lacking."

(d) *Keratosis Obturans.* McGuckin has recently suggested that in many cases the cholesteatoma is a direct result of the accumulation of Keratosis Obturans, a process which Berberich attempted to produce experimentally many years ago.

(e) *Trauma.* Lastly for completeness we note traumatic cholesteatoma which arises following fracture of the temporal bone in which skin is included in the re-united fracture line.

In the writer's opinion the above theories are all open to criticisms even though they all embody the accepted immigration principle. For example acute necrotising cholesteatoma is rarely encountered today and primary enclosed cholesteatoma is almost unknown. Thus these pathological processes can at most only account for a small proportion of the whole. Simple mechanical collapse of the drum is well known, but that of itself is not sufficient to cause epidermosis, whilst the idea of invasion because of unbalanced tissue tension leads us nowhere unless we can explain why the imbalance occurs.

Similarly the theory of accumulating Keratosis leads us nowhere unless we can explain *why* the Keratotic accumulation occurs.

Nevertheless all these ideas are fruitful and in combination yield a plausible theory which not only explains the essential pathological features but leads to positive lines of treatment.

A Synthetic Theory of Epidermosis

We start from the mechanical theory of Bezold. In the course of the upper respiratory infections which are so common in childhood, Eustachian tube obstruction occurs, causing intratympanic vacuum and collapse of the drum head, either in the post superior quadrant, or in Shrapnell's membrane. Dry collapse of the post superior quadrant is a well recognized clinical entity. It is perhaps not so widely recognized that dry collapse of Shrapnell's membrane is *also extremely common.* These two conditions may persist throughout life causing little or no inconvenience. Such ears, however, are vulnerable since under stress they can break down and fall into Epidermosis.

Inflammation occuring in such a vulnerable ear will be *characteristically painless* (or comparatively so) since inflammatory products will seep away through the pre-existing fistula or will ulcerate and burst the atrophic drum without causing the distension of the drum which is the cause of Otalgia in acute inflammation of a normal middle ear.

Moreover, this seeping pus will set up an irritative response in the lining of the fistula leading rapidly to superficial osteitis of the annulus

Pre-Epidermosis

PATHOGENESIS OF EPIDERMOSIS.

Congenital Epiblastic	Metaplastic	Epidermic Immigration	(1) Tubal Obstruction (2) Necrotising Otitis (3) Wachstum Potenz (4) Keratosis Obturans (5) Trauma

The Epiblastic and the Metaplastic theories are to-day regarded as merely theoretical possibilities. The writer, having in the past supported metaplasia, now admits its unimportance and accepts epidermic immigration. This is regarded as a two stage process, i.e. Pre-Epidermosis and Epidermosis.

THE RELATION OF EPIDERMOSIS TO CHOLESTEROL GRANULOMA.

Diffuse Infection of Upper Respiratory Tract in Childhood

↓

Tubal Obstruction

↓

A PATHOLOGICAL SPECTRUM

Mechanical	Mixed Inflammatory and Mechanical	Inflammatory
↓	↓	↓
Collapse of Drumhead (pre-epidermosis)	Exudative Otitis Media	Acute Otitis Media
↓	↓	↓
EPIDERMOSIS	CHOLESTEROL GRANULOMA	CENTRAL PERFORATION

and the formation of granulations. The next stage is determined by the recognized forces of tissue tension. In the normal process of healing, whenever the skin surface is breached, granulations are thrown up and these are covered by ingrowing of the surrounding epithelium. This re-epithelialization is a primordial defence mechanism and its vital importance is revealed by the fact that it is one of the few examples of tissue regeneration found in higher organisms. It is easy to see how this powerful force would urge the hyperplastic epidermic elements of the fistula over the granulations and so into the tortuous spaces of the middle ear, so setting the stage for the final development of Epidermosis.

The key note of this theory is its emphasis on the preceding collapse as an essential preliminary. I regard this as one of the most important pathological conditions within the middle ear, and because of its importance I propose to speak of it as Pre-Epidermosis.

This theory is, in essence little different from the original theory of Bezold and Wittmaack. It is not incompatible with the tissue tension theories providing these are regarded as secondary contributory factors, which only come into action after the establishment of pre-epidermosis.

The conception of epidermosis as a "misplaced healing effort" was put forward by Habermann and is frequently rejected by modern workers who demonstrate how the epidermic elements seem to burrow *under the*

mucosa in a pseudo-neoplastic manner. In reply we merely note that the "healing effort" is responsible merely for the initial entry of the epidermic elements—but once inside the middle ear cleft they are in abnormal conditions and it is not surprising that their behaviour is abnormal. Colman (1960) has produced remarkable experimental proof of this assertion in his studies of grafting at the oval window in the cat. When skin was used it produced the characteristic features of epidermosis in a startling fashion. It proliferated widely, burrowed under the mucosa and finally formed an epidermoid cyst which ruptured into the meatus to form a high postero marginal and attic perforation. This author adds: "There is little reason to doubt that typical cholesteatoma would finally have developed in these animals."

Thus the synthetic theory implies a two stage process

(a) A preliminary mechano-inflammatory process leading to Pre-epidermosis.

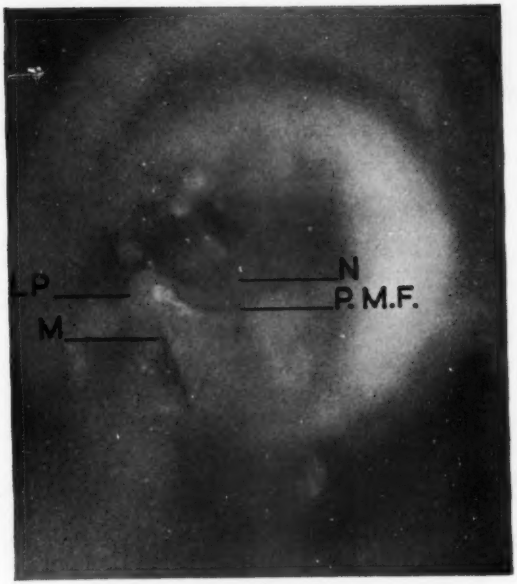
(b) An activating process leading to true epidermosis. This may be local inflammatory in the sense of an accumulation of squames within the epidermic sac or it may be true otitis media.

Evidence for the Synthetic Theory of Epidermosis

The most cogent evidence for this theory is in fact the widespread occurrence of "pre-epidermosis". This can be regularly demonstrated in patients who are entirely unaware of its existence. For example, in almost every case of active unilateral epidermosis the opposite ear will show latent pre-epidermosis. Moreover, cases in this latent condition, if carefully observed over a prolonged period, will be found occasionally to become activated. Conversely, if an active case of epidermosis is encountered at a sufficiently early stage and is treated correctly, the violent response can be cured and the condition returns to quiescent pre-epidermosis.

Again, this theory explains the enigma of the patient who presents with a fully fledged sentinel polyp in Shrapnell's membrane and established attic epidermosis who insists that his trouble only began a few weeks earlier with minimal discomfort and insidious discharge. Clearly this is no acute otitis in the ordinary sense of the word and the Otologist tends to discount the patient's story and to insist that chronic infection must have been present for very much longer than stated. We now see that the truth lies midway. The Otologist is correct in insisting that *some* pathology must have been present for very much longer, i.e. pre-epidermosis laid down probably in childhood. Whilst the patient is correct that the active process only started a few week earlier, i.e. epidermosis.

Lastly, the theory explains how an acute inflammation can *apparently* produce a marginal perforation and a collapsed drum. It says in effect that the collapse preceded the inflammation.



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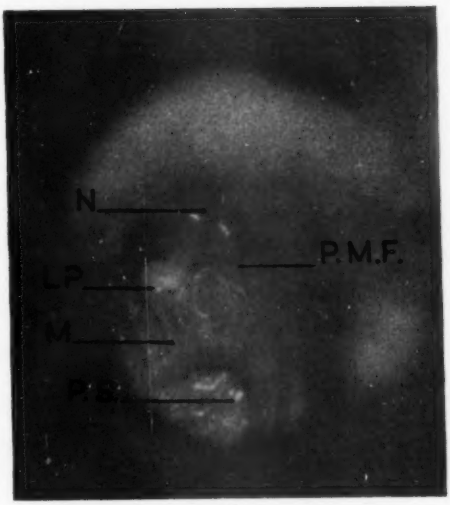


B.

FIG. 3.

PRE-EPIDERMOSIS. THE VULNERABLE EAR.

A shows the characteristic recession of Shrapnell's membrane. The posterior fold of the malleus stands out rigidly. The neck of the malleus can be seen receding into the attic.

B shows pre-epidermosis in *both* sites of election.

LP : Lateral process of malleus.
M : Manubrium.
PMF : Posterior fold of malleus.
PS : Post superior quadrant.

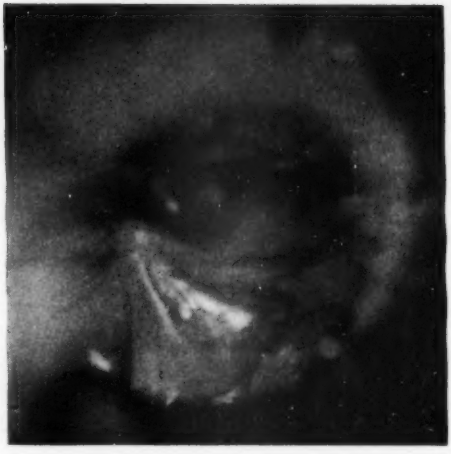
All our understanding of the mechanics of the middle ear indicates that if the drum is normal an acute otitis media must *bulge* the pars tensa and cause a *central* perforation. The post superior collapsed perforation calls for the entirely different explanation outlined above.

Epidermosis and Hypocellularity

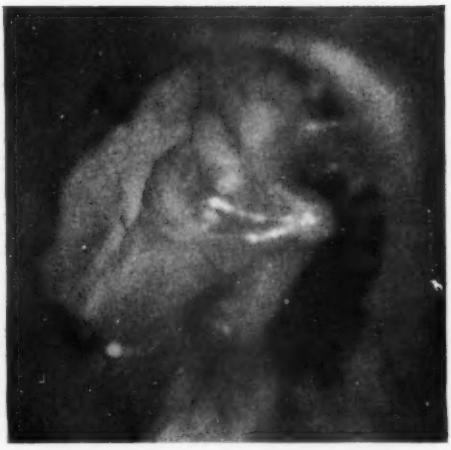
I have shown elsewhere (Tumarkin, 1957) that hypocellularity is a stigma of the upper respiratory diseases of childhood and that it arises as a result of Eustachian obstruction and intra tympanic vacuum. We now see that Epidermosis arises in the same way and we naturally expect the two pathological conditions to occur together. In fact they do so quite commonly, nevertheless it would be wrong to expect that association to be invariable. Pneumatization starts at birth and may be well advanced within a few years. Respiratory infections are commonly delayed till the age of five or thereabouts. Thus a child may easily have developed a large pneumatic system before he develops pre-epidermosis. Indeed our recent investigations suggest that in childhood some degree of pneumatization is almost invariable. Where pneumatization is lacking and the mastoid is truly diploëtic we deduce that pneumatization must have been frustrated at birth as postulated originally by Wittmaack.

On the Nature of Cholesterol Granuloma

I have said that this condition shows no histological resemblance to Epidermosis. The fact remains that Cholesterol Granuloma and Epidermosis are frequently encountered side by side in the same ear, especially in children, and it is natural to inquire whether they may not share some common ætiological factor. In the writer's opinion that common factor is to be found in the familiar syndrome of the diffuse infection of the upper respiratory tract in childhood. In the course of that syndrome the Eustachian tube is very liable to become obstructed. The obstruction may be a purely mechanical phenomenon from adenoid hyperplasia, but more commonly there is an element of salpingitis as well. The final effect on the ear will be determined by the relative magnitude of the two factors, i.e. (1) Mechanical; (2) Inflammatory. When infection is violent acute otitis media develops. When mechanical factors predominate, the middle ear remains dry and the drum collapses. These, however, are merely the extremes of a pathological spectrum. Between them can be found all those varieties of Inflammatory reaction which go to make up the ill defined but vastly important clinical complex "enclosed tympano-mastoiditis". Under that heading I include the various manifestations of exudative otitis media. This condition may sometimes be limited to the middle ear but in most cases it is an exudative salpingo-tympano-mastoiditis. The mastoid air cells become more or less filled with a sullen exudate



A.



B.

FIG. 4.
PRE-EPIDERMOSIS. THE VULNERABLE EAR.

which defies the natural process of absorption and healing, because the obstructed tube will not allow air to re-enter and replace the exudate.

Thus the natural processes of absorption tend to reproduce the original vacuum, the sodden lining of the air cells tends to become vascular, to throw out granulations and to bleed. Thus in most cases of exudative otitis media subjected to mastoid exploration some degree of Cholesterol Granuloma will be found.

Surgical Implications of this Theory

Certain important principles emerge from this analysis.

(a) Exudative tympano-mastoiditis cannot be cured unless tubal function can be restored. Where the condition has gone on to Cholesterol granuloma it becomes necessary—unfortunately—to eradicate the pneumatic system in the mastoid and this calls for a meticulous cortical exenteration. It must be emphasized, however, that the most meticulous mastoid surgery will fail unless tubal function can somehow be restored. Thus, if we divide the auditory cleft into its tubal, tympanic and mastoid segments, each with its individual anatomical, functional and pathological characteristics we see that treatment of Cholesterol Granuloma calls for an attack on the tubal and mastoid segments whilst the tympanic segment should be spared as much as possible. It is true that myringotomy suction is an accepted and valuable line of treatment in exudative otitis media but this is not strictly an attack on the tympanic segment. Its success depends on breaking a vicious circle by removing "glue" and enabling the tube to resume its function, and it must fail if the mastoid segment contains Cholesterol granuloma.

(b) Epidermosis calls for a totally different approach, since attention must be concentrated on the tympanic segment. In the earlier stages it is easy to suck away the tiny sentinel polyp in Shapnell's membrane thus exposing the epidermotic fistula to the desiccation of insufflated Boric iodine powder. In later stages the epidermosis has advanced further into the attic and can only be laid bare by the removal of the outer attic wall by atticotomy.

Provided the mastoid segment is healthy this limited operation can yield gratifying results. For example when, as happens occasionally in adults, the mastoid is "ivory" the simple uncapping of the attic and antrum can yield a tiny healthy cavity within a few weeks (Tumarkin, 1947-1950).

Many writers have maintained (Walsh, 1951) that it is essential to remove the epidermic lining in all cases. This is far from correct. In advanced epidermosis radical surgery is often unavoidable. In the early stages of epidermosis, however, it is not wise to remove the matrix since the process of healing is only too likely to be accompanied by a regrowth of epidermosis into the exposed cavity.

Pre-Epidermosis

Simple Marsupialization and subsequent desiccation will yield satisfactory results but at the price of perpetual vigilance. Patients who have undergone this form of surgery must be supervized regularly for fear of relapse. This is particularly so when the ossicles are thought to be viable and are preserved. The incus is particularly vulnerable and must be closely watched. There is no alternative. Pre-epidermosis can always flare up into Epidermosis.

(c) The combination of Epidermosis and Cholesterol Granuloma is of course the most challenging and implacable of all since all three segments are involved.

Unfortunately this combination is particularly liable to occur in children and the following considerations must be borne in mind.

(1) The disease is not infrequently bilateral.
(2) Even if the opposite ear does not exhibit active pathology it will almost certainly show pre-epidermosis with some loss of hearing.
(3) The infected ear may have reasonably good hearing.
(4) Unless surgery is planned carefully and carried out meticulously it is likely to result in further loss of hearing.

It would seem at first sight that nothing short of radical surgery could suffice and indeed that approach is still extensively used. The functional results of radical surgery are of course notoriously poor, and within recent years, high hopes have been entertained that better results might be achieved by means of the newer techniques of tympanoplasty.

Unfortunately those hopes have to a large extent proved illusory. However successful tympanoplasty may be in other types of tympanomastoid disease it has little to offer in the more florid infections of childhood (Bosatra, 1958).

If there is active disease in the mastoid segment free skin grafts have little chance to survive. Moreover it seems curiously illogical to dissect out laboriously every speck of epidermis only to replace it by means of a free skin graft.

Within recent years, therefore, the author has utilized a combination of

(1) Exenteration of Cholesterol granuloma, plus.
(2) Marsupialization for the epidermosis.

Where the epidermosis has advanced into the antrum destroying the ossicles this combined operation turns out to be nothing other than a classical radical. In earlier cases, however, the posterior meatal wall can be preserved almost intact except in so far as it must be taken down to permit of marsupialization. Providing the cortical exenteration is adequate, the ossicles are viable and tubal function can be restored, it is possible to obtain gratifying results, but the mere statement of these provisos will indicate the enormity of the technical problem, once pathology is well established, and our thoughts naturally turn to the possibilities of prophylaxis. If we cannot *cure* Epidermosis should we not direct more of

our energy towards *preventing* it. In that respect the concept of pre-epidermosis is of critical importance, leading as it does to certain definite lines of action. Our ultimate object is to prevent the development of Pre-epidermosis. This calls for an all out attack on the whole problem of the recurring infections of the childs upper respiratory tract, a problem of the utmost complexity with ramifications into the realms of virology, immunology, climatology, dietetics and so on. The Otologist has the humdrum—but vitally important and deeply satisfying role—of dealing with the ingravescent pathology as it manifests itself in these children often in their earliest years. This work—only too often cursorily carried out in minor ailment clinics is, in the writer's opinion, worthy of far more attention that it has so far received.

It is well known that the dramatic reduction in acute pyogenic ear disease that followed the introduction of anti-biotics has not been accompanied by a comparable diminution in these more chronic diseases. Epidermosis continues to challenge and reproach the Otologist, and it is not surprising that the opinion still lingers in certain quarters that it must be some sort of congenital condition beyond human control. This pessimism is, in the writer's opinion, wholly unjustified. Epidermosis will some day disappear—once we have learnt how to protect the Eustachian tube and control the hyperplastic reactions in the epipharynx. Until that time comes we must set our sights rather lower. Since we cannot —as yet—eliminate pre-epidermosis let us concentrate on preventing its transition to Epidermosis. It is axiomatic that the earlier in the transition we see our patients the easier it is to halt the disease. But since Epidermosis is so insidious cases rarely present themselves in the earliest stages, especially in children. Consequently we must systematically search for them. Routine Otoscopy is already practised together with audiometry by certain Education Authorities but attention is usually directed to major pathology—more especially otorrhœa. We must now intensify this examination with a view to detecting Pre-epidermosis. Any child in whom this condition is detected should be reviewed at close intervals throughout its career and hospitalized at any sign of activity. This applies also to exudative otitis media which, as we have seen, is so closely allied both to Cholesterol Granuloma and pre-epidermosis. Moreover, on reaching maturity the child must be made to understand the potentialities of this menacing stigma so that he may report the earliest sign of efflorescence

The practical vindication of this programme obviously calls for painstaking field work over many years. Its theoretical justification stems from the concept herein expounded of Pre-epidermosis.

Summary

The word Cholesteatoma has outlived its usefulness and should be replaced by *two* terms which must denote unequivocally two quite

different histological entities. Friedmann has suggested Cholesterol Granuloma and Epidermoid Cholesteatoma. The writer proposes Cholesterol Granuloma and Epidermosis.

Cholesterol Granuloma is characterized histologically by Cholesterin crystals, granulation tissue and giant cells. It occurs in advanced exudative otitis media and is common in children as a complication of the diffuse infection of the upper respiratory tract.

Epidermosis is characterized by the presence of true epidermis within the tympano-mastoid cavity.

Various theories of origin are discussed and a synthetic theory is herein proposed embracing them all. This leads to the conception of Pre-epidermosis, viz.: a marginal collapse of the drum either in Shrapnell's membrane or in the post-superior quadrant. This arises—like exudative otitis media—as a result of tubal occlusion and intra tympanic vacuum during the course of the diffuse infection of the upper respiratory tract in childhood. The methods of its transition to active Epidermosis (formerly known as Cholesteatoma) are discussed. Problems of treatment are outlined leading to the following conclusions:

(1) For CHOLESTEROL GRANULOMA—through cortical exenteration plus restoration of tubal function;

(2) For EPIDERMOSIS—marsupialization.

The combination of both conditions presents an extremely difficult problem for which no entirely satisfactory surgical technique has been evolved. At present the author is combining cortical exenteration with Atticotomy. Atticotomy alone is disappointing, so is tympanoplasty. In view of these disappointments the author is now considering the possibility of prophylaxis. True prophylaxis calls for the elimination of the upper respiratory diseases of childhood—a vast problem with no true solution in sight. Meanwhile much can be achieved by the intensive treatment of exudative otitis media. It is suggested that Epidermosis might be controllable if systematic otoscopy of all school children was carried out with a view to compiling a register of Pre-Epidermosis.

All children showing this menacing stigma should be inspected at regular intervals with a view to detecting and arresting the earliest manifestations of Epidermosis. Such a scheme has now been inaugurated in Liverpool and the results will be reported in due course.

The work embodied in this paper has been generously supported by a grant from the Medical Research Council for which grateful acknowledgement is made.

Photographs of the drum head proved far more difficult than anticipated. Fig. 3 is the result of an enormous amount of work by Mr. R. Pracy, F.R.C.S. to whom I record my sincerest thanks. Thanks are due also to Dr. R. W. Rewell who personally prepared Figs. 1 and 2.

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Ear, Nose and Throat Infirmary,
Myrtle Street,
Liverpool

CLINICAL RECORDS

THE OCCURRENCE OF RECRUITMENT IN GLAUCOMA PATIENTS*

By G. CHILARIS and A. COYAS (Athens, Greece)

THE gravity of the disease known among professionals and laymen as glaucoma, is best expressed by Magitot's phrase, "Glaucoma is the number one enemy of the ophthalmologist".

Our intention is not to enter into the, as yet obscure, etiology and pathogenesis of the disease, but we would like to underline that glaucoma is not to be considered as a purely local disease of the eyes. Felix Lagrange has truly made his point in saying that "it is a diseased eye in a diseased body". This aphorism is proved correct not only by clinical fact but also by laboratory experiments, such as the abnormal electroencephalograms obtained by Hartmann (1955) and Duke Elder (1959).

On the basis of the above mentioned concepts we decided to study the existence of disturbances in all the sensorial organs of glaucoma patients. Except in the case of the ear, such a study is fraught through with difficulties as to the unit to be employed in measuring the degree of their functional inadequacy.

Considerable research has been carried out by several authors in this respect and it is because of this that we have decided to undertake this study concerning the occurrence of recruitment in glaucoma patients.

We have been most intrigued by the fact that whereas disturbances of Corti's organ were observed in the majority of people suffering from glaucoma, those complaining of subjective symptoms in their hearing, were very few.

The present study has been restricted to the organ of hearing since there is an anatomical similarity between the ear and the eye. Thus, both contain liquid (aqueous fluid-lymph) which, although differing in consistency, is secreted and drained at a rate which is in immediate relation to the functioning of the corresponding nerve.

Hearing disturbances in those suffering from glaucoma, were reported by Ramboldi as early as 1889. Since then, a small number of authors, have taken particular interest in the problem, among whom a prominent position is occupied by Maglietta (1929), Cordero Porta and (1934), Tille and Leroux-Robert, Worms, etc.

Ferraris de Gaspare and Maffei (1951) believe that Recruitment is an early warning sign of glaucoma, of the same diagnostic value as the provoked hypertension test. Henin (1957) finds the phenomenon of recruitment occurring in about 65 per cent. of simple chronic glaucoma cases and in 10 out of 11 cases of congestive glaucoma.

* Read at the Greek-Yugoslav Congress of Otology, Neurology and Ophthalmology, Beograd, Sept. 1960.

Our own finds

We divided our patients into three categories:

(1) Sixty-two cases, including 48 of simple chronic glaucoma and 14 of congestive glaucoma.

(2) Ten cases of secondary glaucoma.

(3) Eight cases without hypertension, in which the provocation tests proved the existence of hypertension.

The following table shows the results obtained:

Form of glaucoma	Number of cases	Average age	Recruitment	%
Chronic simple glaucoma	48	56	27	56·3
Congestive glaucoma	14	25	11	78·5
Secondary glaucoma	10	49	1 (partial Recruit.)	
Without hypertension. Positive provocation tests.	8	42	3	37·5

It can be seen from the table that Recruitment was observed in 58·5 per cent. of the cases, i.e. in 41 cases out of 70, since the single case of partial Recruitment in the category of secondary glaucoma is considered negligible. The following observations also should be particularly stressed, letting aside the fact that the presence of Recruitment serves as a means for differential diagnosis from presbyacusis, when this phenomenon is absent.

(1) Recruitment was found to be usually homolateral to the affected eye. The contrary, was observed only in 3 cases.

(2) As a rule, the period elapsing between the affection ot the eye and the appearance of Recruitment, could not be determined. Since, however, in three cases of acute glaucoma, Recruitment was almost immediately ascertained, it is believed that the lesions caused in both organs are due to the same pathogenic mechanism.

(3) In 5 cases of surgically treated glaucoma, Recruitment was still present in spite of the normal pressure achieved by the operation.

(4) In congestive glaucoma where disturbances are most evident, there was a greater proportion of cases in which the inner ear was affected.

(5) The audiometric curve was the same in nearly all cases where Recruitment was observed. It fell abruptly in the high frequencies, with regard to both air and bone conduction.

Conclusions

(1) The study of Recruitment in Glaucoma patients shows an increased percentage of cochlear involvement, which is 56·3 per cent. in simple chronic glaucoma cases and 78·5 per cent. in cases of congenital glaucoma. In doubtful cases the presence of Recruitment is probably a warning sign ot glaucoma.

(2) Since: (a) Recruitment and glaucoma were diagnosed almost at the same time and (b) homolateral involvement of the eye and ear was observed in the

main, it is believed that the lesions caused in both organs are due to the same pathogenic mechanism.

(3) All efforts on our part to explain the occurrence of Recruitment in glaucoma patients have so far failed. However, clinical fact shows that both the eye and the ear react similarly to certain neuro-vascular disturbances.

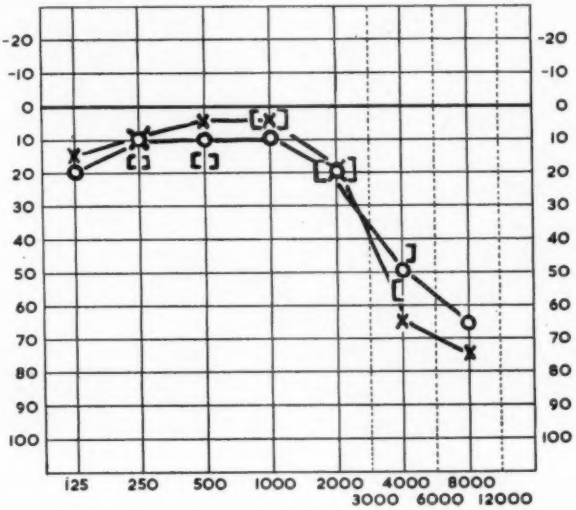

FIG. 1.

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A. Coyas, M.D.
20 Kanari Street,
Athens (K)

EXUDATIVE CHOROIDITIS SECONDARY TO MAXILLARY SINUSITIS WITH REMARKABLE IMPROVEMENT AFTER CALDWELL LUC OPERATION

By K. C. GADRE (Bombay)

MR. N.B.L., a forty-two year old man, railway clerk by occupation, first consulted me on *19.11.59*. He gave the following history: He had high fever, diagnosed as influenza, for a week from *1.8.59*. His cough and cold, however, persisted. On *20.8.59*, when he got up in the morning, he felt he saw flashes of light in front of his eyes. By next day he had a feeling of waves moving in front of his eyes. This persisted for about 10 days. He gradually started losing his eyesight both for near and distance. Images appeared distorted. He felt he was seeing through a cloud and could not identify persons at a distance of about 10 feet. On *8.9.59*, he first consulted an Ophthalmologist, and was prescribed glasses for presbyopia, but these did not help. After persisting with home remedies suggested by lay friends and well-wishers, on *11.11.59* he consulted another Ophthalmologist (Dr. B. Y. Paranjpe). The latter found pigmented precipitates on the posterior surfaces of both the corneae, more in the left eye. Vitreous was full of opacities, and fundi oculi could not be seen. On trans-illumination of the paranasal sinuses he found both the antra of Highmore were opaque. He diagnosed it as a case of Choroiditis, secondary to sinusitis following on an influenzal fever, and asked the patient to consult an Oto-laryngologist.

When I first saw the patient on *19.11.59*, he did not have any symptoms referred to the nose, throat or ears. On physical examination the only positive findings were a polypus seen in the right middle meatus of the nose with muco-purulent discharge in the inferior meatus and the right antrum was tender on pressing. X-rays of the paranasal sinuses showed the right maxillary sinus to be hazy. The other sinuses were normal (Fig. 1 and 2). On *21.11.59*, right antrum puncture was done, and an antral wash was attempted; as there was considerable resistance to the passage of fluid and as the patient started getting pain, the antral wash was abandoned, and it was decided to explore the antrum. On *27.11.59*, the right antrum was opened under local anæsthesia, through the Caldwell Luc approach. It was full of thick pus, and its mucous membrane was hypertrophic and polypoidal. All unhealthy mucous membrane was curetted and an antrostomy was performed in the inferior meatus of the nose. As there was some bleeding, the antrum was packed with roller gauze impregnated with sterile vaseline, and one end of the gauze was brought out through the antrostomy into the nose. Towards the evening of the operation day, the patient reported that he felt his eye-sight was improving. On *29.11.59*, the antral pack was removed, and by this time the patient felt that his eye-sight had almost returned to normal. I was myself very surprised at this remarkable

improvement. On *7.12.59*, he was again examined by the Ophthalmologist who found that the vitreous opacities were much less; the keratitic precipitates were still seen. He prescribed Crooke's Collosal Iodine one tea-spoon twice daily by mouth, and Dionin 1 per cent. eye ointment. On *2.8.60*, that is 8

FIG. 1.

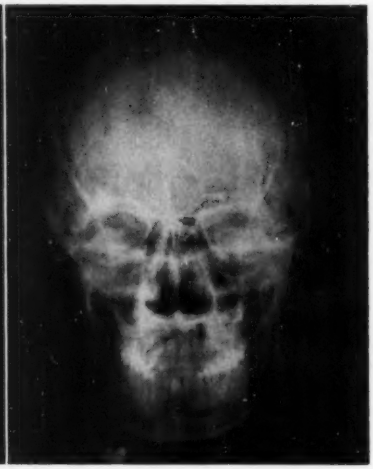

FIG. 2.

months after the operation the patient was again examined by the Ophthalmologist, who found that the vision was normal and the keratitic precipitates and vitreous opacities had disappeared. The patient came to me for a follow-up on *27.11.60* that is 1 year after the operation. He reported that his vision was normal.

Discussion

Sinusitis, especially with retention of pus in the various sinuses, forms one of the less common causes of Uveitis. Septic foci in the teeth and the tonsils are cited as more common causes. Subjective symptoms of Choroiditis are limited to visual phenomena. At the commencement, irritation of the rods and cones gives rise to subjective sensation of flashes of light (Photopsiae). This is frequently followed by a cloud of vitreous opacities indicating outpouring of exudative material. These are the only two first symptoms. When the disease becomes established, due to rucking up of the retina over the affected area, the image becomes distorted (Metamorphopsiae). The clinical course of exudative choroiditis shows usually a sudden onset, marked by a dramatic decrease of vision due to a cloud of vitreous opacities.

The most important element in the treatment of exudative choroiditis is to determine promptly the ætiological factor and to eliminate it. This patient of mine exhibited the text-book symptomatology. His improvement after elimination of sepsis was dramatic.

Summary

A case of exudative choroiditis secondary to maxillary sinusitis is presented. There was a remarkable improvement in the eye condition following a Caldwell Luc operation.

Acknowledgements

I acknowledge with thanks the kind co-operation of Dr. B. Y. Paranjpe, the Ophthalmologist. I thank Dr. S. R. Rege, my chief, for his guidance. I thank the Superintendent of the Lokmanya Tilak Municipal General Hospital, for permitting me to publish this case report.

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Dept. of Otorhinolaryngology,
Lokmanya Tilak Municipal General Hospital,
Sion, Bombay 22, India

MUCOCŒLE OF THE RIGHT MAXILLARY SINUS WITH PROPTOSIS OF THE RIGHT EYE

By L. S. PARKER (Brisbane)

A mucocœle as defined by Schuknecht and Lindsay (1949) is an accumulation of the products of secretion, desquamation and inflammation within a paranasal sinus, with distension of the walls of the sinus. True mucocœles occur typically in the frontal sinus and ethmoid cells and more rarely in the maxillary and sphenoid sinuses. Many described in the literature as those of the maxillary sinus have been instances of a nonsecretory cyst or a dental cyst.

Case History

A 33 years old male was admitted to the Mater Public Hospital with a history of proptosis of the right eye for two months. He had had a lump over the right maxilla for the past 11 years, and this increased in size over the past six months.

He had had an operation on that antrum at the age of 11 years. The nature of this was unknown. He had had nasal polypi removed at yearly intervals for about five years afterwards.

He complained of some blurring of vision of the right eye and some reduplication of distant objects, but he had no true diplopia.

On examination he was a well nourished healthy male. There was asymmetry of the face with a nontender soft swelling of the lateral aspect of the right maxilla. The septum was central, the nasal mucosa was a little hypertrophied and allergic in appearance.

There was proptosis of the right eye with ptosis of the upper lid. Eye movements were full range, the fields of vision appeared full clinically, and there was no diplopia. The Ophthalmologist reported that the proptosis was forwards and upwards, the ptosis was mechanical and produced by restriction of the upper half of lid elevation. The fundus showed pseudo papillœdema with blurring of the nasal aspect of the disc and apical projection of the disc. Exophthalmometer readings were right 2·0 mm. and left 1·6 mm.

The X-ray showed bony destruction of the upper part of the lateral aspect of the right antrum. Both antrums were opaque. Hæmoglobin was 94 per cent. (13·3 grammes per cent.). The Wasserman reaction was negative.

The antrum was explored through the canine fossa and at operation, a large tense cyst filled with greenish yellow fluid was evacuated. The antrum on inspection showed erosion of the antero-lateral wall and the floor of the orbit. The lining membrane appeared atrophic. The propotosis subsided after the contents of the antrum were aspirated. An intranasal antrostomy was performed.

His post operative course was uneventful and there has been no recurrence of symptoms in the past 18 months.

FIG. 1.

FIG. 2.
Radiographs showing erosion of the supero-lateral wall of the right antrum.

There are few reports on the incidence of proptosis in association with mucocœle of the antrum. A report of five cases is noted in extracts from literature in the *Archives of Otolaryngology* referring to report by V. Chinaglia (1951).

There were no signs of operative interference in the antrum, and the operation reported in the history, was probably a proof puncture. There were no polypi in either nostril, and the probable cause was a chronic catarrhal infection with a blocked ostium, producing retention and accumulation of secretion and subsequent atrophy of the mucosal lining from the pressure atrophy which also produced the absorption of bone from the lateral wall of the sinus and floor of the orbit.

Acknowledgements

I would like to thank Mr. Stephen Suggitt for his help in preparing this case report.

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Ballow Chambers,
121 Wickham Terrace,
Brisbane. B.17, Australia

PERFORATION OF THE PALATE SECONDARY TO RHINOLITHIASIS

By V. T. HAMMOND (London)

RHINOLITHIASIS is an uncommon condition and perforation of the palate as a result of a rhinolith appears to be extremely rare. Key Åberg (1921-22) reviewed the literature of 257 rhinoliths. He mentions one case in which there was an associated palatal perforation, but this was thought to be syphilitic in origin.

Polsen (1943) in a review of the literature in 1943 found 396 cases of rhinolith but mentions perforation of the palate only in connection with the case noted by Key-Åberg.

Bovil (1886) reported a case of a giant rhinolith in an Indian, which produced depression of the hard palate as well as facial deformity and VIIth nerve palsy. At operation he noted that there was atrophy of the palatal process of the maxilla on the affected side.

Knight (1903) and Price Brown (1900) mention perforation of the palate as a complication of rhinolithiasis but give no references.

In 1953 Morgan (1957) reported a case in which a perforation of the hard palate was produced by a rhinolith.

Case Report

The patient, a man of 63, was referred to the E.N.T. Department in *April, 1960*. He had first noticed a painless spot on his hard palate, beneath his denture, eighteen months before. For the last three months fluids had been coming back through his nose on drinking. He had had some left nasal discharge for years. Ten years previously a left antral puncture and wash-out had been performed at another hospital.

On examination there was a defect in the hard palate to the left of the midline, and through this a hard black rhinolith could be seen. He wore a full upper denture but the posterior edge of the perforation extended just beyond this (Fig. 1).

On anterior rhinoscopy the rhinolith was completely hidden by a large septal spur projecting to the left, but on passing a probe along the floor of the left nasal fossa, beneath the spur, a hard mass could be felt.

A sinus X-ray showed a little mucosal thickening in the left antrum and an ill-defined opacity in the floor of the left nasal fossa. W.R. and R.P.C.F.T. were negative on two separate occasions. The patient could not recall inserting a foreign body into his nose at any time.

A submucous resection was performed and after removing the bony spur the rhinolith was revealed on the floor of the left nasal cavity. On removal the rhinolith crumbled into several pieces. The mucosa of the floor of the nose

around the perforation was thickened and œdematous, and pieces of this were taken for histological examination. The palatal defect was closed by swinging a broad flap of palatal mucosa across the hole and suturing it to the freshened edges of the perforation. However, this subsequently broke down, leaving the defect unchanged in size.

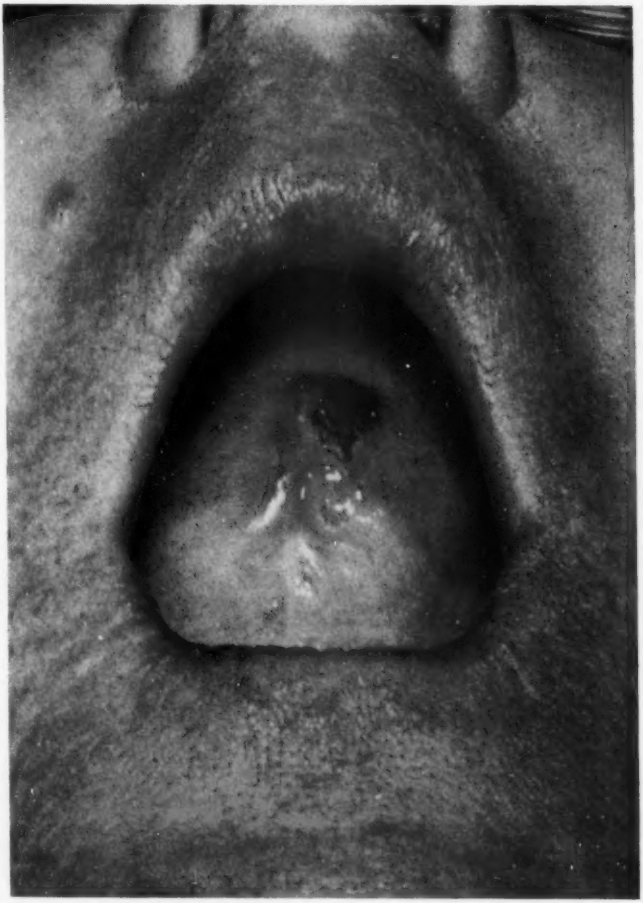

FIG. 1.

The rhinolith can be seen through the perforation in the hard palate.

The pathologist's report on the tissue from the edges of the perforation was "Loose connective tissue infiltrated with inflammatory cells". Examination of the rhinolith failed to reveal any foreign body nucleus in its substance.

The patient has been kept under observation since the time of operation.

The edges of the defect have healed but there has been no reduction in the size of the hole. The upper denture has been extended to cover the hole completely, and when he is wearing this he has no disability.

Discussion

Palatal perforation due to a rhinolith is very rare. Many cases are recorded in the literature in which extensive destruction of the nasal septum and lateral wall of nose has occurred as a result of rhinolithiasis, but perforation of the palate is extremely rare. The size of the rhinolith does not appear to be the important factor in producing a palatal perforation. The largest rhinolith reported (Abu-Jaudeh (1951)) weighed 116 gm. on removal and had eroded the septum and the lateral wall of the nose but produced no palatal deformity. Myerson (1928) removed a rhinolith weighing 56 gm. from a patient who had facial swelling and displacement of the septum but again no palatal deformity.

Morgan suggested that the wearing of an upper denture was a factor in his case, preventing expansion of the palate and so encouraging necrosis. In the case reported here, not only did the patient wear a full upper denture, but in addition the rhinolith was wedged beneath a large septal spur. It seems probable that the perforation of the palate in this case was determined by these two factors.

Summary

An unusual case of rhinolithiasis is presented. The possible factors leading to perforation of the palate are discussed.

Acknowledgements

I would like to thank Mr. G. H. Bateman for allowing me to publish this case and for his advice in the preparation of this paper.

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St. Thomas' Hospital,
London, S.E.1.

METASTATIC CARCINOMA OF THE TEMPORAL BONES

By M. BALSLEV JØRGENSEN (Copenhagen, Denmark)*

ACCORDING to most authors on the subject, malignant tumours rarely affect the temporal bones (Eggston and Wolff, 1947; Marx, 1926). The reported frequency of such tumours, primary as well as metastatic, varies somewhat from author to author. Furstenberg (1924) found the incidence of primary carcinoma in the temporal bones to be 1 in 20,000 of all aural diseases. Lodge, Jones and Smith (1955) found 6 malignant temporal-bone tumours in a population of 1 million over a 3 year period. Metastatic tumours are generally considered to be more uncommon than primary tumours. This is in itself rather strange, since many malignant tumours affecting primarily the breast, prostate, kidneys, and thyroid gland show a marked tendency to metastasize to the bones (Nylén 1936). According to Marx (1926) the metastasization may take place in two ways: (1) By spread of tumour cells with the cerebrospinal fluid to the inner ear as a link in carcinomatous meningitis. This condition Marx named otitis interna carcinomatosa. (2) Hæmatogenous spread with localization in the temporal bones outside of the labyrinth of an origin analogous to that of skeletal metastases in other parts of the body.

Single cases of metastatic temporal-bone tumours have been reported by Nylén (1936), Hamberger and Engström (1941), Lodge, Jones and Smith (1955), Smathers and Vermes (1960) and others.

Owing to their rare occurrence, malignant tumours of the temporal bone often give rise to diagnostic difficulties, particularly in the case of metastases from distant primary tumours. This is exemplified by the following two cases:

Case I

A 67-year-old woman had been suffering from mild diabetes mellitus for 10 years, but had otherwise been fit. One year before admission she developed left-sided middle ear infection with fairly severe hearing loss, which had remained stationary since then, and attacks of shooting pain around the left ear. Four months before admission she had paralysis of the left face on awakening one morning. This was treated by her own doctor by diathermy which resulted in some subjective improvement. About one month after this episode, she began to have increasing rectal tenesmi with frequent imperious defæcation of some mucus and blood. In addition, she was getting ever more tired and lost about 20 kg. The patient was admitted to the University Hospital, Medical Department B, where X-rays of the colon revealed a stenosing tumour at the sigmoid-rectal junction. Operation in Surgical Department C showed peritoneal carcinosis and hepatic metastases, so the procedure was

* From the Department of Otology (Head: Professor H. K. Kristensen, M.D.), Rigshospitalet (The University Hospital) Copenhagen.

restricted to sigmoid colostomy. The patient returned to the medical department where she died 2 months later in a state of cachexia.

Otological examination was performed at about the time of the operation. Otoscopy showed the right ear to be normal. On the left, there was a firm, reddish tumour superiorly in the auditory canal. The tympanic membrane was normal and the mastoid process normal. In addition, there was an almost

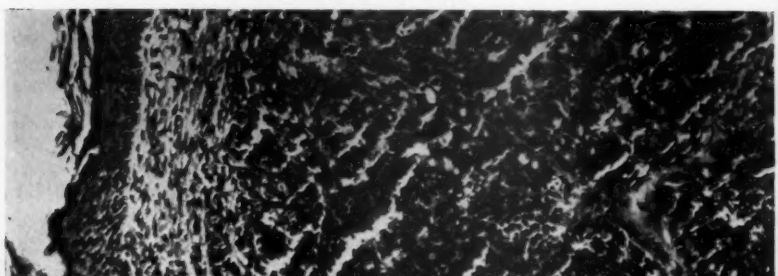

FIG. 1.
Adenocarcinoma in the external auditory canal. × 120. Hæmatoxylin-eosin.

complete left-sided peripheral facial paralysis. Hearing of whispered voice 0·50 m., of spoken voice 1·0 m. Hearing in the right ear normal.

Microscopic examination of a biopsy specimen from the tumour in the external auditory canal showed adenocarcinoma (Fig. 1).

Autopsy was carried out at the University Institute of Pathological Anatomy. It revealed, in addition to the primary tumour, metastases in the regional lymph nodes, the lymph nodes along the aorta and in the hila of the lungs. Moreover, metastases were found in the liver, spleen, ovaries, adrenals, renal pelves, pleurae, and throughout the spine. The skull was opened, and in the petrous part of the left temporal bone, a metastasis, about 3 cm. in diameter, was found. On the right, in approximately a symmetrical site, there was also a metastatic deposit, but only 1 cm. in diameter. No other intracranial metastases.

Case summary: A 67-year-old woman complained of pain around the left ear after an apparently commonplace middle-ear infection. One year later, she

developed left-sided facial paralysis. Not until a few months later did she exhibit symptoms which revealed the primary disease, viz. colonic cancer with multiple metastases. Otological study revealed a tumour in the external auditory canal which a biopsy specimen showed to be adenocarcinoma. Post-mortem examination showed a large metastatic deposit in the left temporal bone and a smaller one on the right. The temporal bones were not examined histologically.

Case II

A man, aged 65, with a history of operation for slipped disc in the lumbar region 12 years previously. For several years he had been having mild dyspeptic complaints, aggravated during the past few months, in the form of dysphagia and a feeling of obstructed passage in the epigastrium. Weight loss of about 20 kg. Moreover, migrating arthralgia. The patient applied to the Ear Clinic because of symptoms from the right ear. For about one year there had been painful swelling behind the ear, mild tinnitus, and slight hearing impairment, but no vertigo. Two weeks prior to admission, he developed severe pain in and around the right ear and at the same time right-sided peripheral facial paralysis. Otoscopy normal on the left. In the right ear the drum was red and swollen, but without defects. In the right mastoid process there was a soft area, about 1 × 2 cm. Lymph nodes on the neck enlarged. Audiogram showed moderate hearing impairment of the conduction type in the right ear, normal hearing on the left. Vestibular examination, including rotatory and caloric test by the Hallpike method did not reveal any abnormalities.

X-rays of the temporal bones revealed slightly reduced pneumatization on both sides with blurring on the right, but no signs of cholesteatoma or other destructions.

Microscopic examination ot excised lymph nodes from the right side of the neck showed metastasis from adenocarcinoma. Supplementary X-rays of the chest and stomach gave rise to a suspicion of diffuse pulmonary carcinosis and gastric ulcer (or tumour?). The case was interpreted as gastric carcinoma with multiple metastases. About one month after admission, the patient died in cachexia.

Post-mortem examination at the University Institute of Pathological Anatomy revealed a bronchogenic adenocarcinoma in the right lung with metastases to the pleurae, adrenals, thyroid gland, hilar lymph nodes, mediastinum, hilum hepatis, and spine. In addition, a metastatic deposit in the right temporal bone. In the stomach a chronic ulcer.

After being fixed in formalin, the temporal bone was sent to our Laboratory, where it was decalcified in equal parts of 8 n formic acid and 1 n sodium formiate. After embedding in celloidin it was cut horizontally into sections of about 20 μ. Every 10th section was stained with hæmatoxylin-eosin.

Histological findings: In the middle ear, effusion containing desquamated epithelial cells and occasional erythrocytes, lymphocytes, and polymorphonuclear leukocytes. Mucous membrane slightly hyperplastic. In the submucosa some hyperæmia of the blood vessels, but no striking accumulation of leukocytes. The appearances were like those seen in serous otitis media. The labyrinthine capsule was normal, without signs of otosclerosis. In the inner ear, apart from

mild autolytic changes, no abnormalities, neither in the cochlea nor in the vestibular organs. In the mastoid process (Fig. 2) the bony tissue was replaced posteriorly by infiltrative tumour tissue with cells arranged partly in formations imitating glandular tissue, i.e. as in adenocarcinoma. Moreover, pronounced congestion of the blood vessels surrounding the facial nerve (Fig. 3). Throughout these blood vessels, tumour cells were observed, singly and in clusters. In a few places, tumour cells were seen also in the intraneural blood vessels.

Case summary: For one year a 65-year-old man had been complaining of a painful swelling behind the right ear, mild tinnitus and hearing impairment. Two weeks before admission, he developed severe pain in and about the ear and at the same time facial paralysis. The primary disease proved to be bronchogenic carcinoma with multiple metastases. Autopsy and histological examination of the temporal bones showed a large metastatic deposit in the right mastoid process as well as numerous emboli of tumour cells in the blood vessels in and around the facial nerve. In the middle ear serous otitis to account for the hearing impairment. No abnormalities of the inner ear.

Comments

In our patients the initial signs of metastases in the temporal bones were pain around the ear, hearing impairment, and facial paralysis. The symptoms in the first case had been interpreted as acute otitis media and later as rheumatic facial paralysis. The second patient ignored his aural pain for a year and did not present himself until massive facial paralysis had developed. In the cases reported by Smathers and Vermes (1960) and by Lodge, Jones and Smith, facial paralysis was also the initial sign. The pain and hearing impairment are explicable by the reactive changes caused by the infiltrative growth of the tumour around the sensory nerves and in the mucous membranes and blood vessels of the cavities. That facial paralysis occurs so early is explained by the fact that the nerve is not protected by a firm bony wall, being in several sites exposed in the pneumatized areas of the temporal bones. As a result, it will soon be reached by infiltrative tumour growth. Metastasization to the temporal bones from more distant primary tumours occurs through the arteries (as illustrated in Fig. 3), and in the bone marrow of the mastoid process tumour cells may be filtered off from the blood stream. Which symptoms or signs occur first depends upon which structures are first penetrated by the tumour tissue. The labyrinth is surrounded by a capsule of firm bony tissue, and it must be assumed, therefore, that symptoms or signs from the inner ear will be fairly late manifestations. And indeed, they were not present in our two cases.

Summary

Two cases of metastatic carcinoma in the temporal bones are reported. Both primary tumours were adenocarcinomas, one bronchogenic and the other one affecting the sigmoid colon. The initial symptoms and signs in both cases were pain, hearing impairment, and paralysis of the facial nerve. The primary tumours were not detected until a late stage at which multiple metastases were already present. Since facial paralysis is a common initial sign of tumours in the temporal bones, this sign calls for painstaking investigation, especially when occurring in patients of the cancer age.

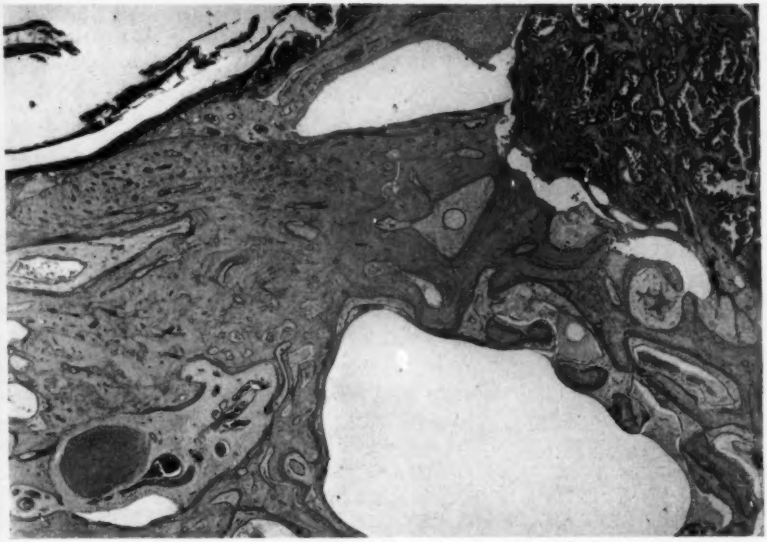

FIG. 2.

Carcinomatous metastasis in the mastoid process superiorly on the right. The facial nerve may be seen in the bottom left-hand corner. × 10. Hæmatoxylin-eosin.

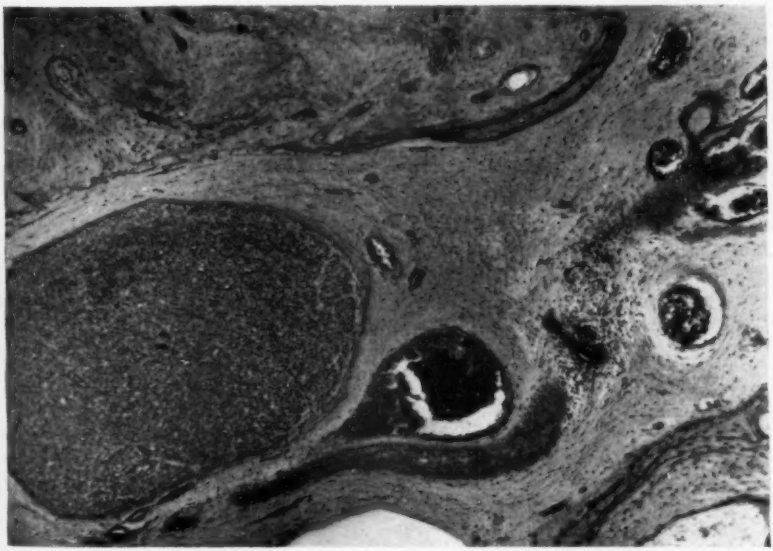

FIG. 3.

The facial nerve with tumour cells in the surrounding vessels. × 45. Hæmatoxylin-eosin.

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E.N.T. Department,
Rigshospitalet,
Copenhagen, Denmark

CLINICAL NOTES

MORE LIGHT ON TRANSILLUMINATION
A REPLY TO MR. TUMARKIN

By M. E. N. SMITH (Swansea)

I am grateful to Mr. Tumarkin for his somewhat personal attack. His eminent name will arrest the eyes of many readers when they would not have been detained by my own, and any reader, knowing Mr. Tumarkin and loving an argument, will rub his hands with glee and read on joyfully. How better could one's thesis be propagated?

Unfortunately Mr. Tumarkin has missed almost completely the point of my paper. As far as I am concerned it is not in dispute that transillumination might, and perhaps frequently does, provide the first clue leading to further investigation and observation and *eventually* the correct diagnosis. But for reasons indicated in my paper it *may not* and, most important point of all, in nearly every neoplastic case where it does, it does so too late to leave the patient with any reasonable hope of cure. The time lag between the original transillumination and the definitive decision that lavage has failed and the time for open exploration for suspected neoplasm has arrived, is surely a matter of weeks and in many places could well be one of months. I cannot recall off-hand any case where such has been the sequence of events i.e. that a neoplasm has been detected at open exploration performed solely because of failure of repeated lavage to produce a clear sinus. Indeed the point of the two cases I quoted was to stress the fact that if diagnosis be missed by failure to X-ray early, and transillumination alone be relied upon, the eventual diagnosis tends to depend upon the appearance of gross and unequivocal evidence of tumour already beyond the confines of the maxilla, rather than upon the exercise of any special clinical acuity in choosing just the right moment to explore. In these respects I speak as I have always found. With Mr. Tumarkin things may be different? Thus my second case is not such a "non sequitur" and is not rendered such by any defect in clinical acumen that may be involved. The X-ray did in fact provide the diagnosis to the seeing eye and the case well demonstrates that X-rays can reveal what transillumination never can—suspicion and near-certainty of neoplasia at a stage in the natural history when all may not be lost.

In this type of argument the truth is often revealed by a single, simple question, the answer to which is no more than an exercise in intellectual honesty. ***How much time can one afford to lose in initiating radical treatment for cancer?***

In this reply I have confined my remarks to the issue of neoplasia as being

the spearhead of Mr. Tumarkin's attack, but without prejudice to my contentions as affecting other forms of sinus pathology. However, I would reiterate that in my opinion the diagnostic obscuration risk is itself of such transcendental importance as alone to justify the consignment of all transilluminating apparatus to either medical museums or dustbins.

London House, Cadle Mill,
Swansea, Glamorgan

INTERNATIONAL SYMPOSIUM ON OTOSCLEROSIS

By I. Simson Hall (Edinburgh)

During the three days from *10th November* to the *12th November, 1960*, a symposium was held in Detroit under the auspices of the Ford Hospital.

A Committee under the Chairmanship of Dr. Harold F. Schuknecht arranged the programme and drew speakers from the United States, Canada and Europe.

There was a large attendance of Otologists, who came by invitation from all over the world including Australia, New Zealand, and South America, many of whom contributed to the discussion on the various points raised by the speakers.

Every aspect of otosclerosis and its treatment was covered, commencing with the anatomy, physiology and pathology, and concluding with treatment by many varieties of surgical techniques.

As the meeting proceeded it became evident that the pooling of knowledge made possible by this symposium was timely. It pin-pointed many difficulties and did a great deal to clear the air of the fog of uncertainty which has surrounded much of the work carried out in this field.

It is evident that we know little of the disease in the treatment of which we spend so much time. The mechanism and physiology of hearing still contain many unsolved problems.

The operations for the relief of otosclerotic deafness both by the older methods and by the new, were fully covered, and a very valuable part of this section was the presentation of the effects of trauma to the vestibule caused during operative interference.

The results of animal experimentation figured prominently in this part of the programme.

Modern methods of stapes surgery were fully covered during the last day, and many methods of treatment were presented with their results, which made an interesting comparison.

Several important points emerged from the Sessions on treatment. It was evident that some universal method of reporting results is required, and one of the greatest obstacles to the agreement of such a method is the inadequacy of our knowledge of the basic physiology which is essential for the establishment of accepted standards.

In the operative methods supported there seemed to be little to choose between the results of the various techniques in the hands of accomplished surgeons. There was general agreement, however, that more time is required for their proper evaluation, and that as yet a sufficient period has not elapsed to decide which of these methods will ultimately emerge as the best treatment.

There seemed little doubt that there is a calculated risk to hearing in stapedectomy, as most surgeons reported a percentage—usually very small—of severely deafened patients. There seemed to be agreement that some cases

were unsuitable for stapes surgery as carrying too great a risk of sensorineural loss; a finding which raises interesting problems in the training of otologists.

It the conclusion is confirmed that certain cases, e.g. those with obliteration of the oval window, are unsuitable for stapedectomy, then our training methods will require review, for few otologists in training are able to perform the classical fenestration operation which is the logical treatment to be carried out by the fully trained otologist.

The Symposium was organized and conducted in a manner which evoked admiration from everyone present and the whole staff of the Ford Hospital are to be congratulated on the success of this outstanding Symposium.

It is understood that J. & A. Churchill Ltd., London, will be responsible for handling in Great Britain the publication of the transactions of the Symposium.

SOCIETIES' PROCEEDINGS

ROYAL SOCIETY OF MEDICINE—SECTION OF OTOLOGY

President: RONALD MACBETH, D.M., F.R.C.S.

Discussion on The Effects of Noise upon Hearing

May 6th, 1960

THE opening papers will be found on pp. 447-486.

WING COMMANDER P. F. KING. In the Royal Air Force we have for some time been aware of the need to protect the hearing of those exposed to hazardous noise levels.

We have acted by defining limits of noise beyond which the hearing is likely to be hazarded and outside which men require protection. In the case of those working on aircraft engines, protection is provided by a British production of the Canadian National Research Council's ear-muff.

We have not yet started the measurement of hearing of men in noise-exposed trades before, during and after employment because of the large staff required to cope with the numbers involved.

We are solving this by constructing an automatic audiometer—the first of three—which will test the hearing of 21 subjects in a 20 minute sitting. This machine, made in the Acoustics Laboratory, is nearly completed, and I had hoped to show it today for your interest.

Two semi-skilled operators only will be required for each machine, to help the subjects and adjust headphones, etc.

After the machine is started, determination of threshold will be made on six frequencies—200, 500, 1,000, 2000, 3,000 and 4,000 c.p.s., in a range of + 50 to —10 decibels, relative to normal threshold.

Test tones will be relayed in descending intensity, in 5 decibel stages, the subject receiving 2 "pips" of the test tone at each intensity. The subject responds by pressing a key, which initiates a photographic process, marking an audiogram form preprinted in the machine.

The performance criteria specified in the British Standard relating to threshold measurement are observed.

With equipment of this sort we aim to measure the hearing of all entries to the Service and to follow all men through their career. Such an audiometer may well be of use in industry, where it could be used on a co-operative basis by several concerns.

D. E. HICKISH, said that the research work on noise at present being undertaken by Dr. Challen and himself at the Occupational Hygiene Service at Slough was an endeavour to study the effect of noise on the hearing of workers engaged

in various processes, particularly those found in small firms. The intention was to perform serial audiometry, at yearly intervals, on a selected group of employees, and to make regular assessments of their noise exposure. One difficulty which had been experienced was the fairly high labour turnover in some of the workshops, which rapidly reduced the size of the group being studied.

Another aspect of their work was the investigation of noise problems on an *ad hoc* basis, both by environmental noise measurements and Temporary Threshold Shift audiometry. It was often relatively easy to show the presence of a hazard to hearing, but extremely difficult to reduce the hazard significantly other than by the use, by the exposed persons, of ear defenders of appropriate type. Many of the processes were inherently noisy, and considerable technical study would be required before noise reduction at source could be effective. However, he felt that there were some basic noisy processes, common to many industries, which merited study, as for example punch presses, automatic machine tools and compressed air jets. He hoped that some appropriate research body might undertake this work for any successful measures could then be widely applied, and make a considerable contribution to the reduction of of occupational hearing loss.

Mr. H. V. Forster said that he had looked forward and had listened with much interest to Dr. Glorig's address, but was no less interested to see how fresh legislation would deal with this problem in Britain.

It was the duty of Otologists to examine the effects of continuous noise upon the Cochlea though its nuisance value was not confined to members of our speciality.

Dr. Donald Stewart's amusing reference to sales resistance to quieter motor bicycles was not unexpected and in his own City when investigating, with the assistance of the Medical Officer of Health, a complaint of a persistant humming noise, not even confined to week days, he Mr. Forster had discovered this to be from a powerful exhaust pump used to protect the worker from silicosis in a factory using sand blasting machinery and at a very considerable distance from the home of those making the complaint.

Dr. G. Kazantzis said Audiometry in industry should not be embarked upon lightly. There are many variables which should be controlled, and factors to be taken into account if the tests are to mean anything. To take but a few:

How long should the subject be removed from a noisy environment before being tested? He should obviously not be tested while under the effects of a noise induced temporary hearing loss.

Extraneous noise within the testing booth should be controlled, and standard maximal noise levels should be adhered to.

The subject should not be tested during the course of an upper respiratory tract infection, and he should be asked about drugs he might be taking—quinine produces a temporary hearing loss.

Again, the subject should understand clearly exactly what it is he has to do, and what signals he has to give.

An experienced operator is required to explain the test, to fit the earphones correctly, to see that friction from the cord does not produce extraneous noise when the signal is being delivered. There are a number of such points to be taken into account.

P. J. R. CHALLEN said, "I would like to comment on certain points mentioned by Dr. Kazantzis because they are of importance in the study of noise and its effect on hearing. Firstly, he is incorrect in his statement that there are no minimum acceptable octave-band noise levels for audiometric rooms, and I would refer him to those, amongst others, drawn up by Webster 1954, Cox 1955 and Glorig and Harris 1957. He poses the question of how long a period of freedom from exposure to noise should be allowed before an audiometric test, so as to eliminate temporary threshold shift. Dr. Hickish and I can speak from personal experience of this problem, having exposed ourselves to various noises for stated periods, and although the question is too complex for a brief reply, we consider that a minimum period of 48 hours should be allowed. However, in practice this is difficult to achieve because, in general, it means that all subjects would be tested on a Monday morning, and in industry this is not feasible. Therefore a compromise between scientific and practical expediency must be a period of 12-14 hours and audiometric tests can then be carried out every morning before entry into the noisy workshop.

Epidemiological surveys of occupational deafness, where the significance of results obtained from serial testing will only be inferred from statistical analysis, demand a greater precision in audiometric technique than is required for the assessment of clinical disorders. Calibration of the audiometer should be carried out weekly by one method or another. (It is essential that all noise measuring equipment be calibrated before each field survey.)

The psychological factors which influence the results of audiometric testing are a subject worthy of detailed study, particularly for the epidemiologist, viz. fatigue in the observer and the observed and the influence of emotional factors and the intelligence level on the response of the observed, etc.

In my opinion, the study of occupational deafness is a neglected one in this country, which is unfortunate because, with the advent of increasing mechanization and automation, noise is a rapidly growing problem. One cannot expect manufacturers to devote time, energy and financial resources to the extremely difficult task of quietening noisy machines if we do not provide them with reliable clinical evidence of the noxious effects of this noise".

REFERENCES

Cox, J. R., Jr. (1955) *Noise Control*, **1**, 25.

GLORIG, A. and HARRIS, J. D. (1957) *In* Handbook of Noise Control, Edited by HARRIS, C. M., London.

WEBSTER, J. C. (1954) *J. Acoust. Soc. Amer.*, **26**, 782.

GENERAL NOTES

VIIème CONGRÈS INTERNATIONAL O.R.L.

Le VIIème Congrès International O.R.L. se tiendra du 23 au 29 juillet 1961 à Paris, à la Nouvelle Faculté de Médecine, Rue des Saints-Pères, sous la présidence de M. le Professeur Aubry.

Trois très importants rapports seront discutés:

1°) Les acquisitions récentes en pathologie et en thérapeutique de l'otosclérose.
Rapporteurs: MM. les Professeurs Sourdille, Sercer and Krmpotic, Weber, Ardouin, Larroude, Ferreri and Schambaugh.

2°) Indications et résultats après 5 ans, de la chirurgie et des associations radio-chirurgicales dans le traitement des cancers du larynx et de l'hypopharynx.
Rapporteurs: MM. les Professeurs Pietrantoni and Agazzi, Alonso, Casadesus, Chevalier-Jackson, Leicher, Leroux-Robert, Ormerod.

3°) Rapports entre l'allergie et les infections du nez et des bronches.
Rapporteurs: MM. les Professeurs Van Dishoeck, Clerici, Hlavacek, Voohorst, Mayer, Laskiewicz, Teracol.

De nombreuses réunions et festivités sont envisagées pour les Congressistes et leurs épouses. A la suite du Congrès, des excursions auront lieu; elles seront proposées dans un avant-programme et réalisées par les soins des Wagon Lits Cook, 22 rue de l'Arcade, Paris, qui sont notre agent officiel habilité pour les transports et réservations d' hôtels.

Pour tous renseignements, écrire au Dr. Henry Guillon, Secrétaire Général, 6 avenue Mac-Mahon, Paris.

TEMPLE UNIVERSITY SCHOOL OF MEDICINE

Philadelphia 40. Pa.

A "Short course in Audiology" is offered several times each year by the Section of Audiology, Department of Otorhinology, Temple University School of Medicine, Philadelphia 40, Pa.

The course will be one week in length and will encompass theoretical and clinical aspects of audition. Hearing testing and interpretation will be stressed. Office procedures, equipment and Industrial Audiometry will be discussed.

Further information may be obtained by writing to Philip E. Rosenberg, Ph.D., Director, Section of Audiology, Temple University Medical Center, 3401 North Broad St., Philadelphia, Pa.

General Notes

INTERNATIONAL SYMPOSIUM ON AUDIOSURGERY

The National School of Medicine and Work, the National Institute of Medicine, and the Centre of Surgical Specialities have organized an International Symposium on Audiosurgery in Madrid in August 1961, on 2nd, 3rd and 4th. The Chairman will be, Dr. Francisco Antoli-Candela.

The subjects to be considered and discussed will be the Audiosurgery of the congenital defects of the ear, the chronic suppurative processes of the ear and its sequelae, and otosclerosis. The surgery of the facial nerve will also be considered. There will be surgical sessions and simultaneous translations.

An official lunch has been arranged and will be included in the subscription to the Symposium. On the 5th August there will be a visit to the "Escorial" and to the "Valle de las Caides", and a "fiesta Campera". A sight seeing programme for the participants and their guests who wish to see the surroundings of Madrid, the South of Spain, and Palma de Mallorca has also been organized.

For further information please write to:

Secretaria General, Dr. A. Juderias, Centro Nacional de Espedialidades Quirurgicas, Ciudad Universitaria, Madrid, Spain.

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THE COLLEGE OF SPEECH THERAPISTS

The College of Speech Therapists is holding a Conference in Birmingham from 24th to 28th July, 1961.

Abnormalities of speech and language will be presented as systemic disorders under the guidance of the concepts of Signs, Signals and Symbols.

For further information please write to the Conference Organizing Secretary, 16 York Road, Birmingham 16.

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BRITISH OTOLARYNGOLOGICAL CONGRESS

It is proposed to hold the first British Otolaryngological Congress in Great Britain in the summer of 1963 and an organizing committee is being set up for the purpose. It is expected that in addition to sessions for formal papers there will be a number of seminars on various topics and revision courses conducted by known authorities.

The Congress will probably last for five or six days.

Further details concerning time and place will be notified in this Journal in due course.

General Notes

SIXTH INTERNATIONAL CONGRESS OF AUDIOLOGY

THE sixth International Congress of Audiology will be held in Leyden, The Netherlands, from September 5th to 8th, 1962.

President: Prof. Dr. H. A. E. van Dishoeck;

Secretary Drs. A. Spoor.

In the program are planned three round-table talks, with associated and free papers.

The subject of the round tables are:

"Frequency analysis of the normal and pathological ear."
Moderator: Prof. Dr. G. von Békésy.
"Central deafness in children."
Moderator: Prof. Dr. J. M. Tato.
"Psychogenic deafness and simulation."
Moderator: Prof. Dr. H. A. E. van Dishoeck.

Official languages of the Congress are: English, French, German and Spanish.
Working languages will be: English and French.

The address of the secretariat is:

Ear-Nose-Throat Department,
Academisch Ziekenhuis,
LEIDEN (The Netherlands).

BACK JOURNALS REQUIRED AND FOR SALE

If any subscriber can spare copies of the January 1945 and June 1950 issues of the Journal of Laryngology & Otology they would be very much appreciated by the Librarian of The National Institute for the Deaf, 105 Gower Street, London, W.C.1.

If anyone would like to buy a set of the journals from 1921 to 1960 (mostly bound volumes), and the two relevant indexes etc., please apply to Donald G. P. Macalister, 5 Clarendon Place, Leamington Spa.

THE MOUNT SINAI HOSPITAL

An intensive postgraduate course in Rhinoplasty, Reconstructive Surgery of the Nasal Septum and Otoplasty will be given January 13, 1962 to January 26, 1962 by Dr. Irving B. Goldman and staff at the Mount Sinai Hospital, New York in affiliation with Columbia University.

Candidates for the course should apply to Registrar for Postgraduate Medical Instruction, The Mount Sinai Hospital, Fifth Avenue and 100th Street, New York 29, N.Y.